THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—28TH YEAR. SYDNEY, SATURDAY, OCTOBER 18, 1941. No. 16.

Table of Contents.

INDUSTRIAL FATIGUE.[1]

By H. M. L. MURRAY,
Melbourne.

RATHER more than two months ago I read a paper on the subject of industrial fatigue to the Melbourne Branch of the Institution of Engineers. On that occasion I spent a good deal of time in talking about elementary physiology, and then concluded with some examples of the application of physiological knowledge to various repetition engineering processes. Tonight I propose to spend very little time on the physiological aspect of the subject, and to devote most of my attention to the discussion of problems of manufacture. I do this for two reasons: firstly, because tonight every member of my audience will have a knowledge of physiology at least equal to, and in most cases far greater than my own; and secondly, because there are certain technical engineering difficulties, a knowledge of which is essential to the comprehension of my subject.

I regard industrial fatigue as a matter of the utmost importance at the present time; unfortunately there is great and widespread ignorance throughout this country, even among those who should make it their business to be well acquainted with the subject. This is rather curious, because there is an immense amount of information available for those who care to look for it—for example, in various publications of the Industrial Health Research Board. And there is nothing new about this knowledge; one of the best pieces of statistical research ever carried out was that of Vernon on output in relation to hours of work in munitions factories in Great Britain during the last war; this was published in the Report of the Health of Munition Workers Committee, 1915-1918. But, although all this knowledge has been accumulated, it appears to be put away in various pigeonholes, and very little use is being made of it. I happen to have access to perhaps more of these pigeonholes than most; and I regard the dissemination of this knowledge as a very important part of my work at the present time.

First, let us try to obtain a clear understanding of what is meant by fatigue, and of the way in which it can be measured and prevented. Fatigue has been defined as "the sum of the results of activity which show themselves in a diminished capacity for work". This definition lays down that fatigue is essentially a negative quality—namely, a diminished capacity for doing work. Fatigue is preceded by activity, and its measure is the diminution in the capacity for work. Bodily sensations, which are often appealed to as an indication of the presence of fatigue, are completely unreliable; fatigue may be progressively advancing without any appreciation by the worker that his activity is decreasing; the steady maintenance of the quality and quantity of his output is the only sure indication that his activity is being maintained. In other words, a diminished capacity for work is the indication that fatigue is present. The problem is to find out how we can establish the existence of a diminished capacity for work; for this purpose some means of measurement must be employed. Fortunately, in most industrial establishments records are kept for business reasons of three factors which are directly applicable to the estimation of the incidence of fatigue. These factors are: (i) output, (ii) lost time and sickness, (iii) labour turnover.

Tonight I intend to confine my attention largely to the first of these; through records of output, the results of activity can be directly measured. However, care must be exercised before deductions are made from output records: the type of work done throughout the period under investigation must not alter; the supply of materials and tools and the pace of machines must not be changed; the output should refer to the same group of workers, and

[1] Read at a meeting of the Victorian Branch of the British Medical Association on September 3, 1941.

these workers must have been employed at the work sufficiently long to have passed through the stage during which practice increases output; and external conditions generally must remain unchanged. In other words, as in any other piece of statistical research, we must as far as possible eliminate the effect of all variables other than that which we intend to study. If these conditions are satisfied, records of output form a most valuable—indeed a most striking—measurement of the incidence of fatigue. This type of record is particularly adapted to the study of the effect of hours of work on the production of fatigue; I shall deal with this in some detail presently.

Before I do so, however, I wish to remind you of the fact that before the last world war very little attention had been paid anywhere to this business of "hours of work". Yet, as a matter of fact, there was at least one well-documented experiment, even then nearly a hundred years old, showing the effect on output of reduction of working hours. In the early decades of the nineteenth century it was quite the usual thing for cotton mill employees to work ninety to one hundred hours per week. There were a few notable exceptions among mill-owners, one of whom, Robert Owen, began in 1816 to run his New Lanark mills for 10·5 hours a day instead of the usual fifteen or sixteen hours. He found that output did not fall below its previous level, although hours of work had been reduced about 45%; there was a great improvement in the cheerfulness of operatives, and it was noticed that they were able to work with renewed vigour.

Owen did not investigate the effect of still shorter hours; but many experiments have been made since his time. Among these may be mentioned the experience of the Scotia Engine Works at Sunderland, where, some time in the eighteen-eighties, hours of work were reduced from fifty-four to forty-eight per week. When the labour costs of the engines manufactured were taken out in detail, it was found that in the first six months after the reduction of hours, these costs had actually decreased. In other words, less working time was being spent on the manufacture of each engine than previously.

A better known experiment was that made by Messrs. Mather and Platt, in 1893, at the Salford Iron Works, a factory engaged in general engineering work. The working week was reduced from fifty-three to forty-eight hours; and when a careful and accurate comparison was made of the output in the year before and the year after the change, it was found that the total production was slightly increased, and that the output of the time-workers was rather more favourably affected than that of the piece-workers. Sir William Mather laid these results before various government departments, and as a direct consequence the hours of work in government factories and workshops were reduced in 1894 from fifty-four to forty-eight per week. There was no falling off in output either by piece-workers or by time-workers.

The lessons of these and other similar experiments were available in the first decade of this century to anyone who had the intelligence to grasp them; but, except in the case of a few enlightened employers, their significance does not appear to have attracted attention. This became very obvious when the war of 1914-1918 broke out. In war industry output and more output is the chief demand; early in 1915 a big increase in the production of munitions in Great Britain became a matter of vital importance. To this end hours of labour were increased, seventy to ninety hours a week being common, and over ninety not infrequent. Of course the assumption was that if one unit of work could be done in one hour, then six units could be done in six hours, twelve in twelve hours, and so on. A simple calculation would give the expected output per week, per month, per year if you like. The actual results were found to belie this assumption, for output did not increase proportionately to time and effort expended. The calculations had gone wrong; the worker had been mistaken for a machine.

Of course nobody can deny that, within certain limits, an increase in the hours of labour will increase output; for example, in six hours more work will be done than in four. The best length of the working day is the problem; if nine hours' work produces nine times as great an output as one hour, will ten, eleven, and twelve hours produce ten, eleven, and twelve times as much?

To deal with this problem and others akin to it, the Health of Munition Workers Committee was set up in Great Britain in 1915. The committee was instructed ". . . to consider and advise on questions of industrial fatigue, hours of labour, and other matters affecting the personal health and physical efficiency of workers in munition factories and workshops". The chairman was Sir George Newman, and other medical members whose names are well known to us were E. L. Collis and Leonard Hill; among the laymen was J. R. Clynes.

This committee, after a very full investigation of the human problems in industry, prepared a report which summed up the experience in munitions factories in the plainest possible terms, which even an accountancy-trained production manager ought to be able to understand. The conclusions reached in that portion of the report which is relevant to my subject tonight may be summarized in two sentences: (i) Except for short periods during an acute emergency, an increase in hours of work, above an optimum which varies slightly in different industries, does not give a proportionate increase in output; on the contrary, it causes the rate of output to fall off with increasing rapidity. (ii) After a continuous period of overtime, improvement in output will take place after the reduction of hours of work; but there will be a time lag, so that this improvement will not be noticed until some time after the reintroduction of shorter hours.

These conclusions were reached as a result of a most interesting series of statistical investigations into the relationship of hours of work and output, investigations which form a classical example of the type of dispassionate scientific inquiry which was so urgently needed at that time. They were compiled originally by H. M. Vernon and published as memoranda of the Health of Munition Workers Committee, and Vernon has given details of them in subsequent publications. Tonight I intend to consider four of these statistical studies in considerable detail. I have chosen these, not because I have any special preference for the work of Vernon, but because they formed the basis for the conclusions of the Health of Munition Workers Committee; and they are published in a convenient form in the report of that committee, of a copy of which I happen to be the official owner. Since the war of 1914-1918 an immense amount of similar work has been done in almost every kind of industry, and in every case the result has been to confirm the conclusions reached in the original investigation.

My first example (Table I) is a study of women turning aluminium fuze bodies. The operatives were standing all day at capstan lathes, and had to subject each fuze body to seven successive boring and cutting operations, requiring close attention throughout, and some delicacy of manipulation, so that no relaxation of effort is possible during the series of operations.

Table I.

Women Turning Fuze Bodies.

Period. (Weeks Ending.)	Actual Hours of Work per Week.	Relative Output per Hour.	Hours of Work × Relative Output.
November 14–December 19 ..	66·2	100	6,620 (=100)
February 27–April 16 ..	53·4	123	6,568 (=99)
May 14–July 2	54·8	134	7,343 (=111)
July 9–July 23 (night shift)	50·0	132	6,600 (=100)
July 30–August 19	47·0	124	5,828 (=89)
August 26–September 1 ..	Holiday for one week.		
September 2–September 23	49·9	135	6,737 (=102)
September 30–October 6 ..	Holiday for four days.		
October 7–November 4 ..	48·3	144	6,955 (=105)
November 11–December 16 ..	45·6	158	7,205 (=109)

In this particular example, as we follow it through from beginning to end over a period of more than a year, three separate factors are at work, each influencing the incidence of fatigue. These are: (*a*) a progressive reduction in

actual hours of work per week; (*b*) complete abolition of Sunday work; (*c*) the granting of holidays, previously withheld.

During the first period, in the weeks from November 14 to December 19, the actual hours of work were 66·2 per week, including eight hours' work on five Sundays out of six; these hours had already been maintained for a long time when Vernon's observations commenced. When hours of work were reduced to an average of 53·4 for the period from February 27 to April 16 the relative hourly output went up to 123, and the total weekly output was reduced by only 1%. During this period there was no Sunday work. It should be explained that the improvement in the output per hour must have been gradual, the figure 123 representing the average over this period. This is emphasized by the fact that the average output per hour during the next period was 134; three Sundays were worked, bringing the hours of work per week to 54·8; the average weekly output was 11% greater than that obtained in the first period. From July 9 to July 23 the operatives went on to night shift, working an average of 50·0 hours per week; the hourly output dropped slightly, probably on account of the disturbance to the normal routine. In the next period of three weeks, when the operatives went back on to day shift, with an average of 47·0 hours per week, there was a startling drop in production. Vernon says that this was probably due to the fact that the operatives had been at work continuously for three months, most of it very hot weather, and had been deprived of the usual Whitsuntide and August bank holidays. I think that this explanation is probably correct, for the operatives were all given a holiday for the last week in August, and during the next four weeks the hourly output rose to 135, which on a basis of 49·9 hours per week gives a weekly output 2% higher than in the first period of this review. At the end of September there was a general holiday of four days for all industry, by government proclamation.

The two periods observed after this date, when actual hours of work were 48·3 and 45·6, show a progressive increase of hourly output to 144 and 158 respectively; these figures are equivalent to a total weekly output 5% and 9% greater than that obtained when the actual hours of work were 66·2. In other words, on this particular job 9% more work was done in 45·6 hours than in 66·2.

My next example (Table II) is one of a job which is done entirely by hand. This is what Vernon calls "sizing a fuze body"; I am not acquainted with this particular name, but I recognized the process at once as soon as I read the description: "In this operation men are engaged in cutting threads in fuze bodies by screwing them into steel taps." (I think that the word "tap" should probably be "die".) This operation requires little manual dexterity, but considerable muscular exertion. Owing to the heavy nature of the work, the men in this particular series of observations always worked in their shirt-sleeves, and usually perspired profusely. This work was always carried out by men; and as it was heavy, the hours worked were no so long as in many other jobs.

Table II.

Men Sizing Fuze Bodies.

Period. (Weeks Ending.)	Actual Hours of Work per Week.	Relative Output per Hour.	Hours of Work × Relative Output.
November 14–December 19 ..	58·2	100	5,820 (=100)
February 27–April 16 ..	50·5	122	6,160 (=106)
May 28–July 16	52·1	119	6,200 (=107)
September 2–September 23 ..	47·6	135	6,426 (=110)
October 7–November 4 ..	51·3	137	7,028 (=121)
November 11–December 16 ..	51·2	139	7,117 (=122)

This table shows that progressive reduction in actual hours of work from 58·2 to 47·6 resulted in an increase of 35% in the relative output per hour, and of 10% in the weekly output. From this stage onwards there is a slight increase in the hours of work, and a continued increase in the weekly output. At first sight the figures in this table appear to contradict each other; I think that the explanation of this apparent contradiction lies in the fact, already mentioned, that after a continuous period of long hours, improvement in output does not commence until some time after the institution of shorter hours. In this particular case the time-lag seems to be pronounced; and it is noticeable that, in the second period, when the hours of work were 50·5 per week, the hourly output was 122, whereas more than eight months later a week in which the actual hours of work were 51·2 gave an output of 139 per hour. I do not think that this table indicates, anywhere, the optimum hours of work for this particular job. Vernon says that there were only two periods where he was observing what he calls an "equilibrium value"—that is, a period unaffected by the time-lag—and those are the first and last of this series of observations. He considers that, with a sufficient length of time to obtain equilibrium, a 47-hour week would have yielded an hourly output of 157, or a total output equivalent to an increase of 27% on that produced by a week of 58·2 hours. Knowing the type of work, I feel certain that better results would be obtained by shorter hours than those observed.

Turning now to a completely different process, we consider an investigation of women machining a screw thread on a fuze body. In this operation the machine does the actual work, and the only function of the operative is to remove the finished product from the machine, place a new article in the chuck, and set the machine off again at the begining of its cycle. This occupies about a fifth of the total time; for the remaining four-fifths the operative stands idly watching her machine. Many people are under the impression that this is an example of an operation in which the machine sets the pace; but the actual figures show that this is not the case (Table III).

Table III.

Women Machining a Thread.

Period. (Weeks Ending.)	Actual Hours of Work per Week.	Relative Output per Hour.	Hours of Work × Relative Output.
November 21–December 19 ..	64·9	100	6,490 (=100)
February 27–April 16 ..	55·4	109	6,039 (=93)
May 28–July 9	54·6	114	6,224 (=96)
July 16–August 6	54·8	121	6,631 (=102)
September 2–September 23	45·5	121	5,506 (=85)
October 14–November 18 ..	48·1	133	6,397 (=99)

Once again we see that a progressive reduction in working hours produces a steady increase in the output per hour and once again the beginning of improvement in output lags behind the actual reduction in hours; this is particularly evident in the last two periods shown in Table III, where early in September a week of 45·5 hours corresponds to an hourly output of 121, whereas six weeks later the output was 133 per hour for a week of 48·1 hours. When we consider that it is possible for the operative to speed up only one-fifth of each cycle of operations, and that the machine really does set the pace for the other four-fifths, it is rather amazing that, when we compare the first period with the last, we should find that when hours of work were reduced by 16·8 per week, total output should be only 1% less.

A similar example occurs in the process which Vernon calls "boring top caps". This name conveys nothing to me; but the job is described as "the light labour of boring top caps on semi-automatic machines", and we are told that the process is largely automatic, and that increase in output can be attained only by a more continuous feeding of the machines. This description enables one to recognize the type of work, if not the actual job. Once again the figures speak for themselves (Table IV).

It will be noticed that in the last three periods the hours of work were for all practical purposes constant; but once again there is a steady increase in the output per hour as the operatives recover from the fatigue produced by the long hours previously worked. The final result, a reduction

in total output of only 3% when hours have been reduced from 72·5 to 54·5, is a striking example of the effect of shorter hours, even in an occupation which is largely automatic and mechanical.

TABLE IV.
Youths Boring Top Caps.

Period. (Weeks Ending.)	Actual Hours of Work per Week.	Relative Output per Hour.	Hours of Work × Relative Output.
November 15–December 19 ..	72·5	100	7,250 (=100)
January 3–February 13 ..	69·1	106	7,325 (=101)
February 21–April 16 ..	54·8	108	5,918 (=82)
May 1–May 28	54·7	117	6,400 (=88)
July 3–September 23 ..	54·5	129	7,030 (=97)

The four examples which I have quoted fall into two groups: (i) those in which substantially the whole cycle of operations can be speeded up by the efforts of the operative (Tables I and II); (ii) those in which only a limited portion of the cycle of operations can be speeded up by the efforts of the operative (Tables III and IV).

If one discusses this matter with a member of the production staff of a large factory, he will usually admit, grudgingly and after much argument, that there may be something in the theory that short hours are worth while on the type of work which falls into the first group. And then his face brightens. "Ah, yes", he says, "all this was very important in the days when most machines were hand-fed and hand-operated; but nowadays most of our machinery is automatic, and the machine sets the pace." It is curious that there should be such a great fascination for accountants and production managers in that phrase about the machine setting the pace. It is one of those quotations which come trippingly off the tongue, and which satisfy the uninquiring mind; but, as I hope to show you presently, to apply this description to all types of machinery used in repetition engineering is entirely fallacious.

It is true that in modern industry there is a great tendency to replace hand work by machines; but these machines range from those which are fully automatic, such as the modern printing press, to those which are power driven, but not automatic at all, such as a foot-operated guillotine. In most machine processes the relative importance of the human and the mechanical element varies between these two extremes; but in practically every case the operator is expected to keep the machine supplied with material. The devices used for this purpose vary enormously; but in general all the semi-automatic machines used in repetition engineering fall into one of three classes. At one end of the scale the raw material is shovelled into hoppers in quantities which keep the machine supplied for long periods; a single worker is therefore able to look after several machines, and in such cases output is assured as long as the machine continues to function in a normal manner, while the operator merely watches a batch of machines and feeds them when necessary. At the other end of the scale each unit of material is fed separately into a hole or division in a moving dial or belt (Figure I).

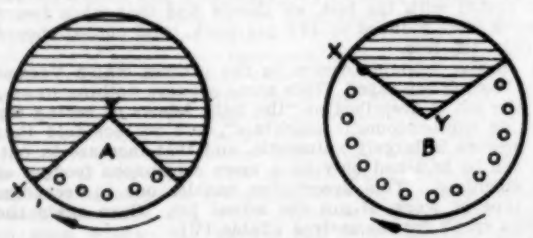

FIGURE I.

In this diagram the unhatched portion of the circle represents a metal disk, rotating in the direction of the arrow; the hatched portion is a cover, hiding the rest of the dial. The articles to be processed in the machine are dropped by the operator into the holes in the dial. It is obvious that this type of work requires some manual dexterity, and a high degree of mental concentration.

Intermediate between the hopper-fed type of automatic machine and various types of dial and belt feed are the large number of varieties of machine in which the feed is by some device such as a chute. Here the operative may not have to feed the articles singly into the chute; it may be possible to use a handful at a time, and there is a varying margin, depending on the length of the chute, between the capacity of the magazine and the demands of the machine. In many cases the articles have to be arranged in a certain way before being fed into the chute; for example, in most bullet machines of this type the bullets have to be arranged with the points in one particular direction. Therefore this work also calls for both manual dexterity and mental concentration.

Now I have frequently been assured by accountants and production managers that in all classes of automatic machinery "the machine sets the pace". A little careful thought will show that as far as output per hour or per day is concerned, this statement can be true only of machinery in which the whole of the feed is completely automatic; it can be applied to hopper-fed machines; but it is certainly not true of belt or dial or chute feeds. The average production engineer is not impressed by a bald statement like that; he likes to see facts bolstered up by figures. Fortunately it is now possible to supply these, for in 1938 the Industrial Health Research Board published a report of an investigation in which machine-feeding processes were studied in great detail.

The earlier stages of this inquiry were devoted to the collection of data concerning a number of different machine-feeding processes. The information obtained included (*a*) the possible output calculated from the machine speed and the hours worked, and (*b*) the actual output obtained from factory records over a period of three months. The machine efficiency is given by expressing the actual output as a percentage of the possible output. I have selected from the records of this investigation figures indicating the machine efficiency in a number of operations in the manufacture of small arms ammunition (Table V).

TABLE V.
Efficiency in Machine-Feeding Processes.

Product.	Process.	Type of Feed.	Number of Machines.	Possible Output.	Machine Efficiency: $\frac{\text{Actual Output}}{\text{Possible Output}} \times 100$
Cartridges.	Heading.	Dial.	14	70	81·2
Cartridges.	Piercing.	Dial.	5	120	57·7
Cartridges.	Choking.	Dial.	4	100	62·7
Cartridges.	Cap former.	Dial.	6	190	66·1
Cartridges.	Forming.	Chute.	6	86	77·4
Cartridges.	Cutting.	Chute.	7	76	76·7
Cartridges.	Necking.	Chute.	5	80	64·2
Cartridges.	Gauging.	Chute.	5	62	78·8
Cartridges.	Assembling.	Chute.	5	114	77·7

It will be observed that in no case is the efficiency of these machines (from the point of view of output) greater than 82%; and in some cases it is as low as 58%. In other words, in these operations the machine has not set the pace. The variation between possible output and actual output is due to one or more of three causes: (i) inefficiency on the part of the operative whose duty it is to feed the machine; (ii) failure of supplies, caused by defective organization; (iii) mechanical breakdowns. Two of these causes depend directly on the presence of the human element in the cycle of operations carried out by these machines; and the avoidance of loss of output by

the third (mechanical breakdown) depends essentially on prompt human action. Therefore, in these cases also the fatigue of the factory worker can and does influence output. To say, as some engineers and most production managers will, that in all types of automatic machinery, no matter what the method of feeding, the machine sets the pace, is patently absurd in the face of the figures which I have quoted.

The case is rather different with machinery in which the feed is completely automatic, for example, the hopper-feed type. Here the variation between possible and actual output is due largely to mechanical breakdown, and hence it is not directly influenced by the fatigue of the operative whose duty it is to feed and to watch the machine. Generally each such operative looks after a group of machines; if the number of machines to be tended and fed approaches the limit of each individual's capacity, then once again the element of personal fatigue is introduced. For example, if it is just possible for one person, working hard, to look after twenty machines for the first three hours of the day, then it is exceedingly doubtful whether the same person will be able to keep all these machines fed to capacity for the last three hours of a twelve-hour shift. In actual fact these conditions do not obtain generally in Australian industry; it would appear that there is still, in general, no dearth of unskilled labour of the machine-minding type, and therefore one does not often see a factory in which operatives have to work to capacity to look after their quota of automatic machines.

I have been unable to find any reference to an investigation into the effect of variations of hours of work upon output per hour in the case of fully automatic machinery; and I think that such an investigation would be a profitless task. But when one turns to records of absenteeism, and of labour wastage, one at once sees the effect of long hours. Absenteeism causes upset of production, because in modern factories, particularly in the manufacture of munitions, the final product is built up of many parts, each the result of one or more operations carried out by separate machines. If the operator of one machine is absent, the orderly flow of parts toward the final assembly is disturbed, and production suffers. Similarly, a high rate of labour turnover means a high proportion of new hands, each of whom has to be taught to operate a machine, thereby occupying the time of a more skilled worker, and so upsetting the flow of production. The relationship between long hours of work and increased absenteeism was rediscovered in England last year. In a recent report by a Select Committee on Parliamentary War Expenditure, there appeared an interesting account of factory experience during the middle of last year:

> After the invasion of Holland and Belgium the Government made a strong appeal for extra work, and factories were working 24 hours a day, 7 days a week. The Whitsun and August bank holidays were cancelled, as were also the local "wakes" customary in some parts of the country. The result at first was a big increase in output. The cry that the country was in danger had a big moral effect, which resulted in a quickening of the stroke. After two months of extreme effort the expected symptoms began to appear. Absenteeism became much more common, and men arrived late for work. Sunday labour led in some instances to men taking a day off in the middle of the week, when of course they only lost an ordinary day's pay, and earned double pay on Sunday to make up for it. In the Royal Ordnance factories absenteeism rose from 10% to 15%, and at the end of July it was decided to discontinue Sunday labour wherever possible, and very often Saturday afternoon as well. The result was immediate. Timekeeping improved and production did not suffer.

If time permitted, I could quote many similar examples from experience in England and in the United States of America during and since the war of 1914-1918. But I think that I have said enough to show that there is ample information on this subject available for all who have the intelligence to use it. Let us see what use is being made of this knowledge.

In Great Britain the general principles are well recognized. There was a big burst of activity in June and July, 1940, when a large section of the community worked twelve-hour shifts for seven days a week. I have already quoted an instance of this. In September, impressed by the experience of the previous months, the Government ordered that the usual holidays should be restored. The Minister for Labour at this time recommended an immediate reduction of working hours to an average of sixty per week, with the aim of a further reduction to fifty-six or fifty-five.

Similarly, in Germany the hours of work have been cut down in consequence of numerous reports that workers in munition factories are showing signs of over-fatigue. *The Times* of December 20, 1939, quoted an order by the German Minister of Labour that the working day must not exceed ten hours.

In Australia "the powers that be" care for none of these things. In this country, when it comes to the organization of shifts, there is a fatal fascination about multiples of the figure "four". If sufficient labour cannot be obtained to keep all the machines in a shop going on three shifts of eight hours, then the only alternative which seems to occur to anyone is two shifts of twelve hours each. If a suggestion is made that two shifts of ten hours might produce a greater output, the only reply is: "But then some of the machines would be idle for four hours a day." It seems quite impossible to push into the comprehension of the average production manager the idea that machines are there to produce goods, and that if, as a result of decreased fatigue of operatives, these machines can be made to produce more goods in a shorter time, then it will pay to have them idle for a few hours a day.

You will recall that I mentioned that machines fall into two groups, those in which substantially the whole cycle of operations can be speeded up by the efforts of the operative, and those in which only a limited portion of the cycle can be speeded up. To these must be added a third group: the machines whose whole operation, including the feed, is automatic. A few weeks ago I walked through a big shop where shells are being manufactured; out of 295 machines in the shop, 85 fall into the first group, 160 into the second, and only 50 into the last. In other words, in 29% of the machines in this shop a reduction in working hours to an optimum will result in an increase both in output per hour and in total output per week. In 54% a reduction in working hours will result in an increased output per hour, and total output per week will remain approximately constant. Only 17% are of the type in which output per hour is not directly affected by the immediate efforts of the operative. And yet every man in that shop was working a twelve-hour shift; and the only breaks in that period are two intervals of twenty minutes each, during which the unfortunate worker is expected to consume and digest a meal. And instead of being deplored as an example of gross inefficiency, this kind of thing is loudly praised by politicians and the Press as a shining example of our magnificent war effort.

If I am asked to comment on this kind of thing, the only printable phrase of which I can think is: "The one thing that we learn from history is that we do not learn from history."

CONTROL OF HEAT LOSS, WITH SPECIAL REFERENCE TO BLANKETS, CLOTHING, EXPOSURE AND RESUSCITATION.

By C. E. Corlette,
Sydney.

Heat loss is loss of calories. People, even medical men, who ought to know better, are accustomed to confuse the terms "temperature" and "heat". Yet it is essential to keep the concepts clear. Heat is energy itself. It is quantitatively measurable by calories, one physiological calorie being the quantity of heat necessary to raise one kilogram of water through 1° C. at a given temperature. On the other hand, temperature is an indication of the concentration of heat, not of its quantity. We can speak of a

degree of temperature, but not of its quantity. The difference is analogous to that between amperage and voltage.

SPECIFIC HEAT.

What is called "specific heat" is a property according to which a substance absorbs a specified quantity of heat per unit of mass in raising its temperature by 1° C. or by any other agreed on range. The "heat capacity" is the heat holdable in a given mass at a given temperature. Water has the highest specific heat, and is taken as the standard, forming unity. Wool has a specific heat of 0·325, cotton 0·32, silk 0·33.[1] The specific heat of the human body will vary to some extent according to the proportions of certain constituents. It has been estimated at about 0·83 by Pembrey.[2] A. C. Burton puts it as between 0·7 and 0·9.[3] If we take 0·83 as the basis of calculation, we find that it would take 2·55 kilograms of wool to equal the heat capacity of one kilogram of the human body. If it were possible to transfer the heat, calculation shows that 175 kilograms (385 pounds) of wool, by falling 1° C. in temperature, could raise the temperature of a man weighing 70 kilograms (11 stone) by 1° C. (1·8° F.). It has been known since the time of Count Rumford that the passage of heat through fibrous stuffs depends more on texture than on the actual material. Loose wool, loose feathers, cotton-wool and felt are the most resistant.[4]

Blankets.

Blankets will approximate in non-conductivity to the materials just mentioned, according to their texture. It is correspondingly difficult to heat any of these non-conductors through and through, and if that were attained, the material would part again with its heat with equal slowness. If a blanket were able to conduct some heat accumulated within itself into the human body, the low heat capacity of the material would make its quantity insignificant.

I have measured the weight, area and volume of many hospital blankets (Sydney Hospital). I find that an old blanket differs little in weight from a new one, but it differs greatly in volume. As an example, a pile of 64 new blankets measured 20 centimetres in thickness, while the same number of old blankets, from which the nap had worn away, measured 12 centimetres. The length was 90 inches (226·6 centimetres) and the breadth 72 inches (182·9 centimetres), giving an area of 45 square feet, or 4·14 square metres, on each side of the blanket. The mean weight of a blanket was three pounds twelve ounces, or 1,700 grammes, with next to no difference between new and old. It is obvious that while nap is important for texture, it contributes very little to weight. The 175 kilograms, or 385 pounds, of wool referred to above equal the weight of 103 Sydney Hospital blankets.

"Warmed" Blankets.

It seems to have been accepted as ordinary common sense, that no one could or would dispute, that in combating shock and heat loss a "warmed" blanket is one of our potent restorative weapons. Nevertheless, let us be critical. Let us now ask how we shall recognize a blanket as a warmed one. In actual practice a judgement is made by feeling it. But what we feel is the surface only, and in a new blanket it is the nap that is touched. In any case, it is the surface of a poor conductor, and if it does feel warm it is no indication of the temperature of the whole. If it is the nap, the total heat capacity of the nap, however hot it is, can amount to nothing more than the product of the specific heat into the mass, and in relation to the whole weight of the blanket the weight of the nap is as nothing.

Let us note next that while one side of a blanket is turned towards the body, the other side is turned away, free to dissipate heat as much in that direction as in the other. The effect of doubling blankets is equal to doubling the weight of a single one.

I suppose nursing practice is much the same in most hospitals. In the Sydney Hospital the tradition is to enclose a patient, immediately after any severe operation, in four blankets, two above and two below. Two of them, those in contact with the patient, are what convention describes as "warm" blankets. No hot tins are allowed anywhere in the bed, a prohibition that should exist in every hospital. On the physical data given above anyone can calculate the maximum calorific value, if blankets are to be regarded as conductors, though if anyone thinks that way, he must also allow for the fact that some part of the blanket covering, and most certainly the outer blankets, will approximate to room temperature, let us say 20° C. This cooler part will demand calories from the warmed blanket, and after that from the human body within, if it is to be warmed up to the temperature of the body surface, which, according to A. C. Burton,[3] approximates a mean of 33° C., at least in the ordinary clothed body. It is easy to calculate requirements, but why waste time on manifest absurdities? That is not the way blankets work.

Texture and Air Content.

If a blanket is classifiable, like loose wool, cotton-wool, feathers and felt, as a bad transmitter of heat, it is to that extent useful as a body covering, able to slow down the outward flow of heat from the skin surface. It is certain that a blanket does act in that way.

Blankets are effective non-conductors in proportion as their texture resembles that of the materials listed above. Essentially, it is not the material "wool", but the quality "woolliness" or "downiness" that gives non-conductivity and gives the blanket the desirable texture. It is on this account that cotton-wool and feathers take close rank with loose wool. There are other qualities that may be desired in blankets. It is claimed on behalf of pure wool that it can maintain its "woolliness" in the presence of a degree of water content (over 30%) that would thoroughly wet and drown other material. Wetness takes away non-conducting quality.

It is to be noted that the most effective materials are relatively bulky for their weight. In the case of loose wool, feathers and cotton-wool the air included has been found to occupy approximately 95% and the solid matter only 5%.[5] The solid material, keratin in wool and feathers, cellulose in cotton-wool, forms a skeletal framework of innumerable fine fibres and the air is enmeshed within this framework. The structure is such that movement of air within the mass is greatly hampered by friction. While the mass is still pervious, the air is held as if by a strong brake.

Transportation of Heat.

Heat can be transported by radiation, by conduction, by convection currents, and indeed by any air current, large or small, whether natural or artificial.

When a patient is covered by a blanket, there is dark body radiation from the skin surface to the opposing blanket surface. Within the intervening space there are also convection currents of air warmed and expanded by direct conduction from the skin, and loaded to or towards saturation with moisture from sweat or insensible perspiration.

The dark radiation, as it strikes the blanket surface, is absorbed, and in that way it heats up the nap. As each fibre warms up, it also warms any air in immediate contact, expanding it and starting a convection current, carrying the heat onwards, where it makes contact with other and cooler fibres and mixes with cooler air, transferring heat until equilibrium is established. The heat and moisture carried by convection from the skin surface accumulates between blanket and body, because of the slowness of its passage through the material. Moreover, when the inner surface of the blanket has been warmed up to skin temperature, dark radiation comes into equilibrium and radiation from the body is balanced until enough heat has passed out through the blanket to alter once more the equilibrium.

Evaporation. Heat of Vaporization.

At this point we turn our attention to evaporation. When water evaporates, heat is absorbed in the process, becoming latent in the vapour, the temperature being unchanged. At 33° C. the latent heat of vaporization of one gramme of water is approximately 580 small calories, or 0·58 of a physiological calorie. Worked out to fluid ounces, it is equivalent to 16·5 physiological calories per ounce. The heat of vaporization must come from somewhere, for without it there can be no vaporization. When water evaporates from the skin surface or from wet clothes the heat will be derived from the body. Under normal conditions a considerable proportion of body heat is got rid of by the evaporation of water from the skin surface. There is no fixed percentage, but at rest, in a respiration calorimeter under room conditions, it has been found to be between 12% and 13% of the total heat output of the body.(7) As soon as skin temperature goes above 33° C., active sweating occurs over the area, and if the area is large, and if the sweat is able to evaporate, the quantity of heat that can leave the body by this corridor is enormously increased. Sweat can be secreted rapidly and in large quantities, at times measurable by pints; but note once more that it is not the mere secretion, but the evaporation, that withdraws heat.

Calculating once more from the case of a man weighing 70 kilograms with a specific heat of 0·83, we find that, unless heat production is increased to make up for it, a loss of 58 Calories will be sufficient to lower body temperature by 1° C., or 1·8° F.—that is, from 98·4° to 96·6° F. These 58 Calories could be withdrawn from the body by the evaporation of 3·5 fluid ounces, or 100 mils, of sweat water, or any water wetting the skin. Heat production can be depressed by blood loss, by anæsthetics, or by other means, but when metabolism is normal, heat loss is balanced by heat production. Sudden, excessive and uncontrolled heat loss will upset equilibrium. So comes a "chill", with whatever results that may imply.

Radiation.

Heat energy is emitted by radiation from bodies containing it. The rays pass readily through pure air, but not through water or fog. When rays cannot pass through, they are absorbed, or if not absorbed they are reflected. Dark non-reflecting substances absorb the heat, becoming thereby warm, and in their turn they radiate away the heat they have absorbed. The warmer the surface, the more heat it radiates; the colder the surface, the less it radiates.

Heat radiates away from the surface of the body. According to circumstances, it radiates into outer space, into walls and furniture of a room, or into the clothing, if the body is clothed. In return, the body receives heat radiated from bodies in space, from the room, or from the clothing. The balance may show loss or gain.

Because a warmer surface radiates more than a colder, a flushed skin radiates away proportionately more heat than a cold and relatively bloodless one, and so, when it is important to conserve heat, a cold skin is protective. A dose of alcohol can make a cold skin flush and feel warm; but with that there can be an increase of heat loss by radiation and conduction.

Conduction.

Heat conduction means, basically, direct transference of energy from contiguous molecules to contiguous molecules by collision. In transference by convection there is mass movement in addition to molecular movement, and the substance must be in the gaseous or liquid state. Molecules whose energy has been increased by direct conduction diffuse away and are replaced by others in which the process is continuously repeated. In the whole mass the outcome is an increase of volume. With increase of volume there is a decrease of relative density. A convection current is thus set up, carrying with it the energized molecules, which in turn transfer energy as they collide with other molecules, wherever the current takes them.

Exposure to Cold, Wet and Wind.

With a wet uncovered body, evaporation will go on so long as the adjacent air is able to take up vapour—that is, until it reaches saturation point. Then it must stop. That is possible when the atmosphere is stagnant, but if it is moving, the vapour passes on with it. Heat loss continues.

With a dry uncovered body, heat, as well as being radiated, is conducted into the air adjacent until that adjacent stratum is warmed up to skin temperature. Equilibrium is then established. But if there is movement, the warmed air is swept away and replaced by unwarmed air. Heat loss then continues.

If the wetness of a wet skin comes from sweating, the cooling produced by evaporation will at length bring the skin temperature down below 33° C. When that occurs, active sweating will cease, and that particular channel of heat loss is closed. But if wetness continues, loss of heat by vaporization will continue to temperatures indefinitely low, so long as the environing air remains unsaturated. The controlling factor is the vapour pressure.

The thinner the fabric, the more open it is, and the less it is associated with any nap, the nearer do conditions approximate to those of nakedness, and the less is the restraint on conduction and convection heat loss. Direct radiation is absorbed into the fabric, but heat so absorbed will pass easily through it and radiate beyond, or be carried beyond by conduction and convection.

When clothing is wet, it contains water in the liquid state. In the state of vapour or gas, molecules are approximately ten times as far apart as they are in the liquid state. It follows that there are about a thousand times more molecules in a cubic centimetre of liquid than in the same volume of vapour. In the case of water larger figures are given. The weight increases correspondingly. At the same time, the heat capacity, volume for volume, is enormously increased. For these reasons liquid water can receive and transmit many more units of heat per second than the same volume of air. Liquid water also absorbs radiated heat, and because conduction and convection to the outside are so much easier when the interstices of the fabric are occupied by water, the temperature on the inner aspect of the clothing will rise comparatively little. Thus there can be but little reciprocated radiation. The radiated heat goes, but it does not come.

This information about the condition of wetness is not all we shall discover, but it is relevant. Add to it now the information already gained about the calories absorbed in the vaporization of water, 0·58 of a large calorie per gramme, or 16·5 calories per fluid ounce. Add to it also the remark, already made, that in the evaporation of water from wet clothes the heat of vaporization will be derived from the body. We shall gain some further information when we consider wind effects.

Body Coverings.

If the body covering includes a layer of impermeable or nearly impermeable material, such as leather, oilskin or rubberized fabric, convection currents, whether of air or water, cannot pass through. The atmosphere beneath the impermeable layer becomes vapour-saturated, so that sweat is not evaporable, and remains liquid. Therefore calorie loss by evaporation is prevented. Heat can still be transmitted, passing into the layer by molecular collision (conduction) and then beyond by convection or radiation, according to opportunity. If the outer surface of the impermeable layer is naked, as in a leather coat or mackintosh, it will be exposed to the influence of wind or any air currents on that side, and in addition there will be free radiation. With such a covering as a fur coat, escape of heat on the exterior is slowed down by the furry layer. If there is a leather, or other impermeable exterior, with a non-conducting lining or other clothing beneath, only such heat will reach the outer layer as can pass through the material interposed between it and the body, the source of the heat.

Wind Effect.

We have now to consider wind, or similar air movement, in relation to heat transference. Convection currents have already been discussed. Wind is like convection on a grand scale, complicated by the rotatory movement of the earth. Between the two extremes comes the household draught. Winds and draughts vary in temperature, in dryness, in barometric pressure, and in velocity. The kinetic energy, or power of doing work, depends in great part on the velocity of the current. A rise in barometric pressure has the same effect as a rise in velocity, so in the making of calculations a correction has to be made to standard pressure.

A permeable fabric offers a frictional resistance to perflation that varies with its thickness and structure. A strong wind will have sufficient kinetic energy to overcome much friction, and in that degree will be able to pass through the material. It will thus make more intimate contact with the skin surface, which is, in effect, the same as stripping off the clothes and reducing the body more or less to nakedness.

More intimate knowledge of wind character can be obtained. If we care to take the trouble, we can calculate the heat-lifting (or refrigerating) capacity of any air current, for any proposed level of temperature, for example at 33° C., the approximate mean surface temperature of the clothed human body—that is, provided we have the following data: temperature, relative humidity, barometric pressure and velocity. I have done this with a number of winds, calculating the heat-lifting potentiality as large calories per cubic metre per second, at a standard pressure of 1,000 millibars. The figures obtained are not conjectures, but measurements. Their accuracy depends on the accuracy of the meteorological observations.

We shall obtain a better objective conception of the wide differences in the qualities of wind currents by some actual comparisons.

The first on the list is a tropical hurricane from Cairns, February 9, 1927: temperature, 76° F.; humidity, 95%; barometric pressure, 29·150 inches; velocity, 80 miles per hour; heat-absorbing capacity at 33° C. and 1,000 millibars pressure, 84 Calories (or units) per second by conduction, 328 by vaporization, a total of 412 per second.

The second is from Sydney, at 12 noon on December 4, 1929: direction, east; temperature, 77·5° F.; humidity, 80%; barometric pressure, 29·521 inches; velocity, two miles per hour; heat-absorbing capacity, two Calories per second by conduction, 10 Calories by vaporization, a total of 12 per second. Looking at these figures, we find it easy to understand why people feel so cold during a hurricane and yet so hot when there is no wind. It is not the temperature of the wind that makes the difference, but its capacity for removing heat.

Similar measurements of a few more winds will be relevant. The third is from the same day's record as the second, but at 3 p.m., only three hours later. The direction had changed from east to west-north-west; temperature, 91·2° F.; humidity, 26%; barometric pressure, 29·402 inches; velocity, 30 miles per hour; heat-absorbing capacity by conduction, −0·4, by vaporization 212, a total of 212 Calories per cubic metre per second. In this case the wind is much hotter, and yet its capacity for removing heat is between seventeen and eighteen times as great as it was three hours earlier at a lower temperature. This is brought about wholly by vaporization capacity, assisted by velocity. There was no cooling by conduction. Conduction of heat had even begun to reverse its direction. Note the negative sign.

The fourth is still from the same day's record as the second and third, but later, at 9 p.m. The wind had once more changed direction and was blowing strongly south-south-west. It was in fact a typical example of what in Sydney is called a "southerly buster". The temperature was now down to 68° F.; humidity, 81%; barometric pressure, 29·473 inches; velocity, 35 miles per hour; heat-absorbing capacity by conduction 57, by vaporization 218, a total of 275 per cubic metre per second.

The fifth is a dry winter wind in Sydney, at 3 p.m. on June 9, 1929; direction, west; temperature, 61° F.; humidity, 34%; barometric pressure, 29·748 inches; velocity, 30 miles per hour; heat-absorbing capacity by conduction 65, by vaporization 268, a total of 333.

The sixth is a winter wind, said to be a "Chinook", from Edmonton, capital of Alberta, Canada, 2,187 feet above sea level, at 8 p.m., on January 4, 1911: direction, west; temperature, 38° F.; humidity, 81%; barometric pressure, 27·25 inches; velocity, 36 miles per hour; heat-absorbing capacity by conduction 130, by vaporization 306, a total of 436. The reader noting that humidity was 81% must remember that this was at a low temperature. A humidity of 81% at 91° F. would carry between five and six times the weight of water carried at 81% at 38° F. A Chinook wind is described as relatively mild in temperature, at least for that part of the world, being above freezing point, and thawing snow. But this one, at least, had considerable velocity, and although above freezing point, it could remove much heat from an unprotected human body in a very short time.

In the study of these winds we find much that is relevant to our purpose. For example, we can now view with better understanding the effect of any kind of windbreak, a wall, a tent, a rude hut, a closed chamber, or the regulation of doors and windows. And again we bend our attention with better understanding to the consideration of clothing, bedding, and so on. As we have already seen, these more personal coverings have several functions, but they do not cease to be wind-breaks by being other things as well.

In every wind here examined the potential vaporization factor has greatly exceeded that of conduction. But in the cooler climates, and especially in the winter season, the conduction factor is the one that matters most. The vaporization factor is then quite secondary. In hot weather the conditions are reversed. However, at temperatures below the sweating point there is always an insensible perspiration through the skin. It is greatest in the hands and feet, and it increases as temperature rises;[6] but in ordinary circumstances it is probable that vaporization does not assume serious importance until there is sweat to vaporize, and that does not occur until the critical point for sweating is reached, at or near the skin temperature of 33° C. (91° F.). In cases of shock and syncopal collapse this mechanism can be upset, and in such circumstances the body may break out into a cold sweat, whereby comes a special opportunity for heat loss by vaporization.

Protection against Wind Velocity.

Take now the case of an injured and shocked person lying out exposed in wet clothes to a wind such as the first, fourth, fifth or sixth studied. In such a case the vaporization factor is added to the conduction factor, and all the Calories removed must come from the body. Or take the case of a person emerging hot, flushed and sweating from a theatre or dance room into an outdoors dominated by one of the same winds. The result in all must be a flood of heat loss, depending for its quantity and severity on the duration and extent of exposure, and on the heat-lifting capacity of the wind, which we can now express in units. What shall we do?

Unquestionably and imperatively, the first thing to do is to counter the wind. Let us reduce the velocity, and for the effect to be gained by this compare the first wind studied with the second. Contrive a wind-break. For the person coming out of a hot theatre we can say that to avoid a flood of heat loss he should put on an overcoat able to meet the emergency, checking perflation, and so checking conduction and evaporation. For a shocked person in wet clothes the best wind-break will be contrived by wrapping him up in an impermeable covering such as a mackintosh cloth. This will at once stop all perflation and all evaporation, and it will be all the better if outside it there is a blanket to block exit of conducted heat. The damp or wet clothes will be warmed up by body heat to the temperature of the skin surface, involving a heat loss of one Calorie per kilogram or litre (35 fluid

ounces) of water present in the clothing for each degree rise in the Centigrade scale, or 1·8° F. That will be unavoidable, but the total thus lost will be trifling in comparison with vaporization and conduction losses incurred by exposure. It will be much the same as the heat lost by having a drink of cold water.

The principles of protection continue. There may be no longer the exhaustion and lowered metabolism of a long anæsthesia. But there is loss to make good by some means. The first and most important means is the vitality still remaining in the organism. We must take every possible care to conserve all that is left, and when that care is given, metabolism can revive and heat can once more accumulate.

In our study of wool and blankets we learnt that they tend to bring into being a body environment consisting of a layer of air at a temperature of about 33° C. and saturated for that temperature with aqueous vapour, a tropical environment, and this environing air is still, except for its intrinsic convection currents. If we have such a layer intimately surrounding the body and remaining there continuously, it will block completely the loss by vaporization, and diminish very greatly the loss by conduction and radiation. If the patient is closely wrapped in such a covering, the quantity of air filling the space between blankets and body will be small, and the warming up and moistening of this small quantity of air will abstract only an inconsiderable amount of heat. In the absence of wind or draught it will not be necessary to interpose the further protection of a mackintosh, but there is no reason against it.

Resuscitation Chambers.

Without adding any extraneous heat we could use a closed chamber as a shield against the outward passage of heat and accumulate body heat, as in the case of a blanket wrap. But in that case there would be an initial loss to overcome, by reason of the warming up and the vapour saturation required for the air within the space. If we know the volume of the chamber, we can calculate the heat required to bring the air up to 33° C. and to saturate it at that temperature with the vapour, beginning with any initial temperature and vapour content. The space would probably measure less than a cubic metre; but for convenience of calculation we can suppose it to be that size. Let us calculate it first as for cold air, taken in at 0° C. and saturated with all the vapour it can hold at that temperature. We find that it takes 10·016 Calories to warm one cubic metre of air from 0° C. to 33° C., and it takes 21·157 Calories to saturate at 33° C. one cubic metre of air that was already saturated at 0° C. The weight of water thus taken up is 36·47 grammes, or 1·28 ounces. Let us now calculate the same problem for room air, taken into the chamber at 20° C. (68° F.) and at 50% of saturation, a comfortable indoor air condition. We find that this would take 3·67 Calories to warm to 33° C. and 17·223 to vaporize enough water to saturate it. This water would weigh 29·69 grammes, or 1·04 ounces. The loss occasioned by using the colder sample of air totals 31·17 Calories, enough to lower the temperature of a man weighing 70 kilograms by 0·536° C., or 0·96° F. If we used the warmer air of the living room, the total heat required to warm and moisten it would be 20·893 Calories, about a third less, with a correspondingly smaller demand on the body. If the volume of the chamber amounted to only half a cubic metre, all the figures would be halved. These calculations also explain why, when outside heat is not conveniently and safely available, it is well to adopt close wrapping instead of a chamber or tent.

Warming and Humidifying from Without. Incubator System.

We can prevent the drain of heat if we warm and humidify the air of the chamber by other means, derived from without. It is possible even to do more, and transfer some heat to the body. We can do this by incubator equipment. A unit is attached that is able to pour in a sufficiently warm and sufficiently moist stream of air. A much higher humidity would be advisable than that (60%) used in an egg incubator, but the temperature (100° F.) need not, and probably should not, be higher. What would seem to be a suitable heating-humidifying device, of reasonably low cost, has been described by G. Wishart and A. B. Baird.[3]

A warm and dry air current impinging on a dry skin surface that is relatively cool, and below 33° C., could transfer heat to it by conduction. It would not be quite so efficient as a fully moist current, equally warm, because there is some insensible skin perspiration below the sweating point. As soon as surface temperature rises to 33° C. the transfer of heat to the body is stopped automatically by the outbreak of sweating. This would in ordinary circumstances cause loss of heat through vaporization. But if the air is already saturated with moisture, the sweat cannot evaporate, and transfer of heat from air to body can go on as long as the air is warmer than the skin. The sweat then bathing the skin will simply remain there, taking no heat for vaporization. If the lining of the chamber is warm, dark body radiation will be returned in exchange for what comes to it.

Radiator System.

Another type of chamber has been employed, depending primarily on radiation from electric filaments. Heat rays pass through air without warming it. If the air becomes warmer, it is secondary to contact with surfaces heated by the rays. The rays warm only surfaces directly exposed to them. If the air becomes moist, it is through evaporation of sweat from the skin heated by the radiation. However, if the air is relatively cold, it cannot hold as much water vapour as it would at higher temperatures.

When resuscitation chambers of either type are used, it would seem reasonable to have the patient naked, or nearly naked, especially when radiation is used. Blankets will prevent heat gain just as much as in other circumstances they can prevent heat loss.

As regards the choice between incubator and radiator type, experience is required. The fact that a radiation chamber is more easily improvised should not count for too much. I think the incubator type is likely to be the better one.

Questions.

There are questions that arise in connexion with these warming chambers. For one thing, how far should we go in warming the body surface? It is known that a warm bath can produce syncope in some people, apparently by an increased flow of blood to the surface causing a decrease in intracardiac pressure. In shock there is already an accumulation of blood in the viscera, and there may have been blood loss, all detrimental to blood pressure. Perhaps blood drawn to the skin may come from the visceral reservoir, but I do not know.

Another question relates to sweating. Can we be sure that if we induce sweating, the patient can spare the water lost? It is quite likely that in some cases he cannot. In any case, let him drink.

Alcohol.

Turn now to alcohol. We have seen that it can warm and flush the skin. If this occurs when the surface is exposed to wind and cold, the result will be a flood of heat loss. However, alcohol is completely oxidized in the body, with production of 7·1 calories per gramme consumed, exactly the same as when it is burned in a lamp. Now if the channels of heat loss are sufficiently blocked when alcohol is given, there can be a gain of 7·1 calories per gramme, and no loss. Whisky and brandy contain approximately 36% of alcohol, weight in volume. Therefore one fluid ounce contains 10·2 grammes, yielding 72·5 Calories. If the specific heat of the body be taken as 0·83, these 72·5 Calories can raise the temperature of a man weighing 70 kilograms by 1·25° C., or 2·25° F. We noted earlier that it took 31·17 Calories to warm to 33°

C. and to moisten to saturation a chamber containing one cubic metre of air, starting at 0° C. and saturated at that temperature. If that heat had to be drawn from the body, we could compensate for the loss by consuming 206 minims, or about 3·4 drachms, of whisky or brandy.

Respiration Loss.

In considering atmospheric environment, we have up to this point taken notice of the skin surface only. Turn now to the heat lost by respiration. How far is it important to control the inspired air? A man lying at complete rest at near basal metabolism will breathe approximately 500 litres per hour, equal to one cubic metre per two hours, at ordinary sea-level pressures. Breathing air at 0° C., saturated at that temperature, and at 1,000 millibars pressure, he will require, for each cubic metre respired, 8·477 Calories to warm it, and 18·056 to moisten it to saturation at 33° C., the temperature of respired air. The water removed from the body by moistening the air will weigh 31·13 grammes. The total heat removed per cubic metre respired will be 26·523 Calories. If, instead of the cold air, he breathes air at room temperature, say at 20° C., and at 50% humidity, the requirement for warming will be 3·339 Calories, and for moistening, 5·593, vaporizing 9·64 grammes of water. The total heat required will be 8·932 Calories.

Let us translate these figures from the respiratory tract into fuel values of whisky or brandy. Calculation shows that the 26·533 Calories removed from the body by breathing the cold air for two hours equal the fuel value of 175 minims, or just under three drachms of whisky. The 8·932 Calories removed by breathing room air equal the fuel value of 52 minims of whisky.

Instead of using values of alcoholic fuel, we could have used that of carbohydrate. One gramme of sugar yields 4·2 Calories. A square tablet of cane sugar weighs exactly five grammes and yields 21 Calories. Anyone can pass on from this to express all the calories given in preceding calculations in terms of carbohydrate consumption. There is valuable fuel in sweetened tea.

Warning.

If we have learnt successfully to conserve body heat and to restore a depleted supply, let us not fail to appreciate the possibility that what is potent to give is potent to go on giving, till what is good has passed over into what is dangerous. Hyperthermia is at least as dangerous and as much to be avoided as hypothermia. No patient in a resuscitation chamber should be left. He should be under constant and careful watch. He should not be kept in it too long. In an efficient apparatus it would be easy to kill people.

Summary.

The specific heat of a blanket, as compared with the human body, is small, and its relative heat capacity is altogether puny. As to "warmed" blankets, one may feel warm and yet be cold, because the nap only is warm. If a blanket were really warmed through, it would not readily give up its heat, because it is a bad conductor. But that is just the real value of a blanket, giving it the property of slowing down loss of heat. By this effect it keeps the air around the body warm and humid. Others materials have other special values. Mackintosh and oilskin not only keep off wet, but, worn over wet clothes, they can limit vaporization and loss of the latent heat of vaporization. It is possible to calculate the heat-absorbing capacity of wind, in relation to the body, stating conduction and vaporization values separately. The factor of controlling importance is the velocity. In one example the heat-lifting capacity was seventeen times more than that of another wind of lower temperature, but of small velocity. A large part of resuscitation consists in the blocking of heat loss. Therefore control of air currents is of first importance. Next comes provision of warm air as an environment. If the environing air has to be warmed up by heat derived from the body, the smaller the air space to be warmed, the better. The heat and moisture required to warm and humidify any resuscitation chamber can be calculated, and if it has to be provided from the body we can find at what cost. Heat is not easily transferable from surrounding air to the body, but it is possible to some extent if the skin temperature is below 33° C. (91° F.). On occasion, alcohol might assist in heat production, since when oxidized within the body it yields 7·1 calories per gramme. Sugar might also be useful, contributing 4·2 calories per gramme. The heat lost by respiration of air at any temperature can be calculated at the amount per cubic metre respired. Examples show that the potentialities of heat loss by this channel are much less serious than those to which the skin surface is liable.

Note on Calculations.

Nineteen years ago, while working at a paper "On Climate, Weather, and Fat Covering in Relation to Metabolism", published in The Medical Journal of Australia of February 17, 1923, I needed certain information. There was no such information to be had. The necessary work had never been done. There are quantitative characters in different masses of air that are not noted in official meteorological observations. They elude direct observation. But they make our environment. In the past we have sensed these different properties, but in a vague way. Before we can record and compare such things we must be able to measure them, and be able to write down the measurements in figures—that is, in simple mathematical form. There appeared to be no practical system of doing this, or if the means existed, they were out of reach. I thought it could be done, but saw it as a most formidable undertaking. I hoped the same idea would come to someone else with more time or better opportunities. But as the years passed and nothing happened, I was driven to it. It was time-consuming and difficult, but it was done. The greater part of the calculated matter in the present paper has been made possible through the tables constructed in that work, and for practical purposes there is no other way.

But unfortunately the papers have never been published, and I do not expect them to be published in my lifetime, if ever. No journal publishes material of the kind. Some institutions do it, but only when it is work produced by members of their staff. However, I hope, after making a further revision, to deposit copies of the papers concerned in the Public Library of New South Wales. There are three papers by me, with the following titles: "On the Heat-Absorbing, or Heat-Lifting, Capacity of Wind in Relation to the Human Body. With a Table for Calculating it." "Measurement of the Drying Capacity of Wind." "On the Calculation of the Heat Dissipated from the Body by Respiration. With a Table Designed to make Calculation Easy at any Temperature, any Humidity, and any Pressure of the Air." I hope to be able to get permission for a fourth, not written by me, namely, "Note on a Diagram for the Calculation of the Heat-Lifting Capacity of a Cubic Metre of Air, Based on the Tables of Dr. C. E. Corlette", by W. H. H. Gibson, lecturer in mechanical engineering at the University of Sydney.

References.

(1) Dietz: Quoted by J. Schofield: "Cloth Finishing", 1927, page 372.

(2) M. S. Pembrey: Schaefer's "Text-Book of Physiology", Volume I, 1898, page 839.

(3) A. C. Burton: "The Specific Heat of the Human Body", *The Journal of Nutrition*, Volume IX, 1935, page 277.

(4) "International Critical Tables", Volume II, page 239.

(5) A. C. Burton: "The Average Temperature of the Tissues of the Body", *The Journal of Nutrition*, Volume IX, 1935, page 261.

(6) "International Critical Tables", *loco citato*.

(7) F. G. Benedict: "The Influence of Inanition on Metabolism", Carnegie Institute of Washington Publication No. 77, 1907, page 477.

(8) F. G. Benedict and H. H. Wardlaw: "Some Factors Determining the Insensible Perspiration of Man", *Archives of Internal Medicine*, Volume XLIX, 1932, page 1119.

(9) G. Wishart and A. B. Baird: "A Heating-Humidifying Device for Incubators", *Science*, New Series, Volume LXXIV, 1931, page 269.

BLOOD PRESSURE CHANGES FOLLOWING LOCALIZED MYOCARDIAL DEATH.

By T. E. Lowe, M.D., M.R.C.P., F.R.A.C.P.,

Acting Senior Lecturer in Pathology, University of Melbourne; Honorary Physician to Out-Patients, Saint Vincent's Hospital, Melbourne.

(From the Pathology Department, University of Melbourne.)

For many years experiments have been carried out on mammalian hearts in attempts to produce lesions similar in effect to those of myocardial infarction in man. In these experiments muscle death has been caused by the tying off of branches of the coronary arteries (a method used by many workers), by the introduction of radon seeds (Haney *et alii*, 1933), by cautery burns (Abramson *et alii*, 1936), and by the application or injection of electrolytes (Kisch *et alii*, 1940). With none of these methods is the lesion strictly comparable with myocardial infarction in the human being. Vessel ligation in animals is not associated with atheromatous changes so universally seen in the human coronary arteries in myocardial infarction; the use of radon produces slow muscle death, and the cautery an extensive lesion, and the application of electrolytes produces only transient changes in the muscle.

These methods have been used to study such different aspects of the effects of local myocardial death as changes in systemic blood pressure and changes in the electrocardiogram. Different experimenters have interpreted their results in ways which are not readily compatible with one another.

The present experiments are concerned with the immediate effects on systemic blood pressure of localized muscle death in the ventricles. In these experiments small amounts of a concentrated solution of phenol in water were injected into the ventricular walls of dogs' hearts. These injections produce rapid localized areas of muscle death without interfering with the vascular arrangements of the ventricular muscle. The extent of the muscle death can be controlled by the volume of injected fluid, and its localized nature enables a description of the lesion to be made in terms of the muscle bundles involved. In addition, it is possible to study the effect of these sudden muscle lesions on the general circulation during the first few minutes after damage, uncomplicated by any appreciable vascular disturbance in the coronary circulation.

Method.

In these experiments dogs were anæsthetized with chloroform after a preliminary injection of morphine sulphate. An intratracheal tube was then inserted in the neck and connected to an artificial respiration pump and the anæsthetic agent was changed to ether. A cannula was inserted into a common carotid artery and connected to a manometer recording on a smoked drum.

The thorax was opened through the anterior wall by the removal of the anterior half of two ribs. The site of the ribs removed varied in different experiments, according to whether the apex or the base of the ventricles was to be exposed. The pericardium was approached transpleurally and opened on the anterior surface. The cut edges were then fixed to the edges of the wound by sutures, so that the heart lay in a bed with fixed relations to the mediastinal structures in spite of the movements of manipulation.

After exposure of the heart, a concentrated solution of phenol was injected into a selected region of the ventricular musculature through a fine needle attached to a tuberculin syringe of one cubic centimetre capacity, the heart being controlled with the fingers.

At the finish of the experiment the animal was killed, the heart was sectioned, and the site of the damaged area (Figure I), easily visible to the naked eye, was determined. In some cases microscopic sections were made also.

Results.

The extent of the necrosis produced by the phenol is indicated diagrammatically in Figure II. In this diagram the areas indicated represent the actual transverse extent of the naked eye lesion; their longitudinal extent varied from six to eight millimetres.

A summary of the experiments performed is presented in tabular form (Table I), the site of injection being related with the changes in blood pressures observed.

The sites of injection were mainly through the anterior and lateral surfaces of the ventricles, with the exception of 6*b*, which was through the posterior surface of the heart. The necrotic areas produced each involve portion of one or more of the various ventricular muscles, but there is nothing to suggest that one particular muscle is of more importance than another in the maintenance of systemic blood pressure.

Figure I.

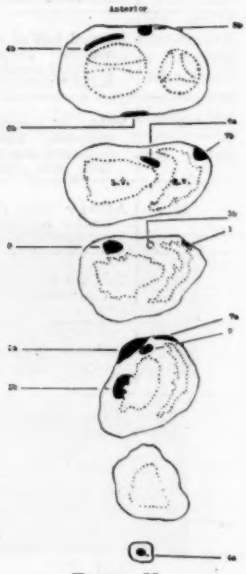

Figure II.

Figure I.—Photographs of two sections of the heart in Experiment 2. The areas of necrosis are indicated by the arrows.

Figure II.—Diagram illustrating the sites of muscle damage in the various experiments. The sites of the two fatal injections are indicated in outline only. The other sites are shown in solid black. The drawings represent transverse sections of the ventricles at levels passing from the base at the top of the figure to the apex at the bottom.

A study of the kymograph tracings (Figures IIIA and IIIB) shows that only in the case of injections 1 and 3*b* was there any sustained fall of blood pressure during the time of observation, which extended in several cases to over thirty minutes. In Experiment 1 (as the blood pressure was steadily falling before injection and fell no faster afterwards) it is probable that the animal was dying before the phenol injection was made. In Experiment 3*b* it appears that death was due to the injection, which produced a necrotic area in the anterior end of the interventricular septum approximately half-way from the base to the apex of the ventricles. It will be noticed that in many of the tracings there is a transient fall of blood pressure at the time of injection. This is due to manipulation of the heart itself. A similar fall is observed with the control needling of the ventricle (tracings 4, 5 and 6). It will also be noticed that simple needling of the ventricular muscle produces no change of blood pressure lasting more than a few moments, and the change is no greater than that produced by manipulation of the ventricles.

Table I.

Experiment Number.	Site of Injection and Muscle Involved.	Systolic and Diastolic Blood Pressure. (Millimetres of Mercury.)		Duration of Experiment after First Injection.	Tracing.	Comments.
		Before Injection.	After Injection.			
1	Antero-lateral wall of right ventricle half-way from base to apex, not involving pericardium or endocardium. Scroll or D.S.S.[3] muscle.	76/64	38/36 (3 minutes).	3½ minutes.	1 (↓)	Phenol, 0·3 cubic centimetre, injected. Death 3½ minutes after injection. Blood pressure had been steadily falling from 126/104 from beginning of experiment. Effect of muscle destruction on blood pressure doubtful.
2a	External half of anterior wall of left ventricle at junction of lower and middle third, pericardium involved. S.S.S.[1] muscle.	66/46	68/58 (8 minutes).	—	2 (1st ↓)	Muscle destruction had no effect on blood pressure.
2b	Internal half of lateral wall of left ventricle involving portion of papillary muscle and endocardium. S.S.S.[3] muscle.	68/58	96/90 (23 minutes).	33 minutes.	2 (2nd ↓)	Combined effects of two sites of muscle damage produced no fall of blood pressure.
3a	Middle of anterior surface of left ventricle, but only apparent damage being done to pericardium from some spilt carbolic.	72/68	132/124 (7 minutes).	—	3 (1st ↓)	Fluctuations in blood pressure before second injection during manipulation of heart.
3b	Anterior end of interventricular septum not involving either pericardium or endocardium.	132/124	64/60 (2 minutes).	19½ minutes.	3 (2nd ↓).	Death 12½ minutes after second injection.
4a	Extreme apex of left ventricle below ventricular cavity.	92/80	92/80 (1 minute).	—	4 (1st ↓).	Transient fall of blood pressure following injection.
4b	Wide area in anterior wall near base of left ventricle.	120/104	106/96 (9 minutes).	18 minutes.	4 (2nd ↓).	Muscle damage produced only slight fall of blood pressure.
5a	Injection of 0·05 cubic centimetre of carbolic attempted, but no evidence of any muscle damage.	—	—	—	5 (1st ↓).	
5b	Anterior wall of left ventricle almost at base. S.S.S. muscle.	58/56	62/60 (7 minutes).	15½ minutes.	5 (2nd ↓).	No fall of blood pressure.
6a	Interventricular septum at junction of upper and middle thirds near the left ventricle but not involving the endocardium. D.B.S.[4] muscle.	80/74	70/64 (6½ minutes).	—	6 (1st ↓).	Fall in blood pressure just before injection due to manipulation of heart. See initial control needling.
6b	Superficial muscle on posterior wall of left ventricle near intraventricular septum. Pericardium involved.	70/64	64/60 (6½ minutes).	—	6 (2nd ↓).	Slight drop of blood pressure.
6c	Injection attempted into lateral wall of right ventricle, but may have gone into ventricle; no evidence of muscle damage.	64/60	24/22 (1 minute).	30 minutes.	6 (3rd ↓).	Blood pressure rose to 68/62 in six minutes and 74/68 at seventeen minutes. Only transient fall of blood pressure.
7a	Anterior end of intraventricular septum, involving only superficial muscle at junction of middle lower thirds.	90/84	88/84 (12½ minutes).	—	7 (1st ↓).	Blood pressure rose to 92/86 in eleven minutes. Transient fall only.
7b	Antero-lateral wall of right ventricle, involving surface and centre two-thirds of total wall. At junction of upper middle thirds. Involving S.S.S. and D.S.S. muscles.	92/86	92/86 (4 minutes).	15 minutes.	7 (2nd ↓).	Muscle damage produced no fall of blood pressure.
8	Anterior wall of left ventricle, junction of upper and middle thirds. No involvement of superficial muscles. D.B.S. or scroll muscle.	90/84	96/90 (10 minutes).	10 minutes.	8 (↓).	Muscle damage produced no fall of blood pressure.
9	Anterior wall of left ventricle junction of middle and lower thirds. Not involving superficial muscle. D.B.S. or scroll muscle.	80/64	86/64 (12½ minutes).	32 minutes.	9 (↓).	Muscle damage produced no fall of blood pressure.

[1] S.S.S. = Superficial sino-spiral muscle.
[2] S.B.S. = Superficial bulbo-spiral muscle.
[3] D.S.S. = Deep sino-spiral muscle.
[4] D.B.S. = Deep bulbo-spiral muscle.

Apart from these changes in blood pressure, the destruction of the areas of muscle concerned produced no immediate change in systemic arterial blood pressure. Changes in pulse pressure were not investigated, but are roughly indicated by the thickness of the kymograph tracing. The heart rate was not recorded.

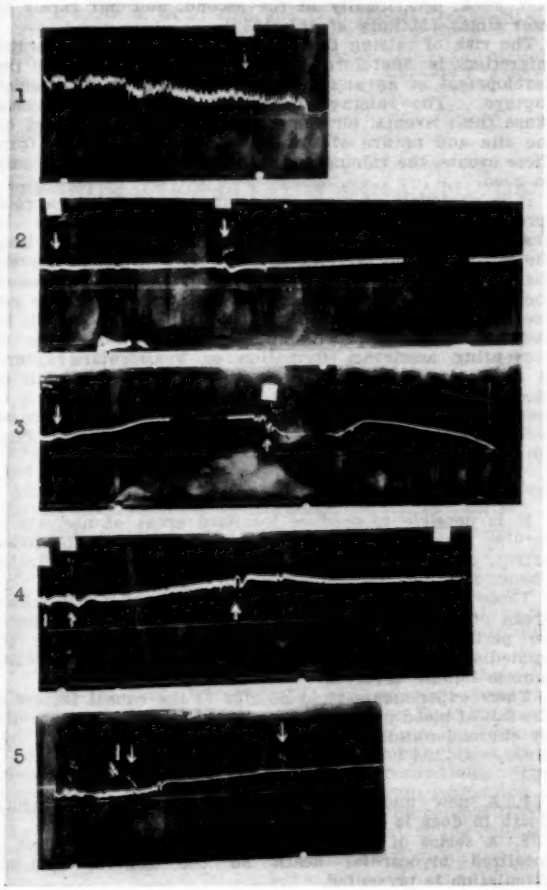

Figure IIIa.

Photograph of kymograph tracings of the blood pressure values recorded in the various experiments. The middle line indicates the zero blood pressure. The lower line is the time marker (graduations are visible in only a few tracings). The arrows indicate the time of injection of phenol and the dashes the time of control needling.

Discussion.

The various sites of muscle damage produced in these experiments include some portion of each of the muscle bundles of the ventricular wall, both superficial and deep, but with the exception of Experiments 1 and 3*b* the damage produced no pronounced or sustained fall of blood pressure. It seems doubtful whether the muscle damage from the phenol injected was responsible for the death in Experiment 1, for the blood pressure was falling steadily before the injection was made and fell no faster afterwards. These observations are in accordance with those of de Waart (1936), who found that there was no fall of arterial blood pressure in monkeys for at least twenty-four hours after the tying of branches of the coronary arteries. They do not, however, agree with the results of Robb (1935), who tied off the branches of the coronary arteries in dogs and came to the conclusion that there was a large fall of blood pressure when the deep muscles were put out of action, and very little fall when the superficial muscles were damaged. Gross (1938), however, found that there was a delay of twenty-four hours before the blood pressure fell after ligation of branches of the coronary arteries.

As a basis for the treatment of myocardial infarction in human beings, it is important to determine the cause of the fall of blood pressure so generally recognized to occur after myocardial infarction, even if the fall is delayed for some hours after the onset of the condition. The fall of systemic blood pressure leads inevitably to a diminished coronary blood flow, and as was shown in a previous paper (Lowe, 1941), this exaggerates the damage to the myocardium in an atheromatous circulation in which an infarct has occurred, for a good pressure difference across anastomotic channels is essential if adequate anastomotic channels are to function.

It is a well-known observation that in laboratory animals when the systemic blood pressure falls a vicious circle is set up, in which a diminishing coronary blood flow and so diminution of cardiac activity occur, leading quite rapidly to death unless the circle is broken, as, for example, by the administration of adrenaline. Similarly, among human beings there are cases in which patients who have been in the habit of taking amyl nitrite to relieve angina of effort and have taken the drug after an undoubted infarction, have rapidly died; death was due presumably to the fall of general blood pressure produced, with concomitant failure of the coronary circulation.

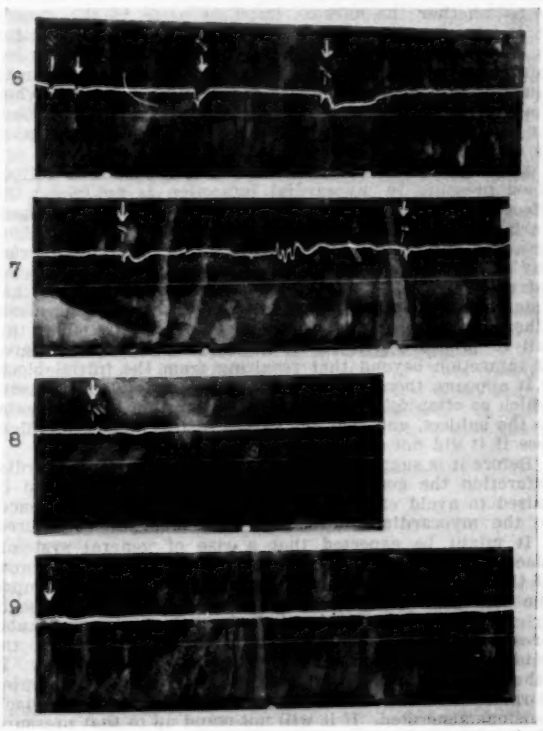

Figure IIIb.

Photograph of kymograph tracings of the blood pressure values recorded in the various experiments. The middle line indicates the zero blood pressure. The lower line is the time marker (graduations are visible in only a few tracings). The arrows indicate the time of injection of phenol and the dashes the time of control needling.

An analysis of the possible factors causing this fall of blood pressure, and of the risks of raising the blood pressure artificially after it has fallen, will serve to place the problem of the treatment of myocardial infarction in perspective, and will possibly suggest lines of treatment for patients suffering from myocardial infarction.

There are three ways in which myocardial infarction may lower the systemic blood pressure: by the quantity of muscle destroyed, insufficient being left to maintain cardiac output; by the liberation into the general circulation of some vasodilating substance; or by a nervous reflex mechanism.

Robb has maintained that the important factors are the quantity of muscle destroyed and the particular muscle bundle involved. If this were the sole cause, one would expect the fall of blood pressure to occur rapidly after the actual destruction of the muscle tissue, and this is not borne out by the experiments here recorded, nor by those of de Waart or Gross; moreover, clinically it is recognized that the fall of pressure does not always occur at the onset of myocardial infarction. Further, it does not seem to have been established clinically that involvement of large portions of the deep ventricular muscles is always followed by a considerable fall of blood pressure (Lowe, 1939; clinical record of Case 27).

Bennet and Drury (1931) and Kellaway and Trethewie (1940) have shown that a cardio-depressant vasodilator substance is liberated from damaged heart muscle. There is, however, present in the muscle an adequate amount of enzyme to destroy this substance, so that it probably does not reach the general circulation. It would of course be of considerable value in the establishment of anastomotic channels, but of little effect on the circulation in general.

The part played by nervous reflexes is not clear, for there is a great difference of opinion between workers as to whether the afferent impulses arise in the vessels or in the damaged muscle (Wiggers, 1937). Clinically, in many, but by no means in all, cases of myocardial infarction severe pain is present at the onset and may cause a state of shock, which is greatly diminished when the pain is relieved by the breaking of the reflex path—for example, by removal of ganglia or by their destruction by alcohol (Coates, 1940).

Although the mechanism causing the sustained fall of blood pressure in myocardial infarction is not clear, the effect of the fall on the coronary circulation is obvious. Such a fall leads to a drop of the coronary arterial blood pressure, and so to a fall in the pressure differences across any anastomotic channels that may be available for the infarcted region (Lowe, 1941). If the infarction has occurred in a system in which there is gross generalized atheromatous blockage of the coronary vessels, then this fall of pressure will result in an extension of the area of infarction beyond that resulting from the initial block.

It appears, then, that the fall of systemic blood pressure which so often follows myocardial infarction is detrimental to the subject, and that the extent of the infarct would be less if it did not occur.

Before it is suggested that in the treatment of myocardial infarction the general systemic blood pressure should be raised to avoid extension of the infarct, the consequences to the myocardium of such a rise must be considered.

It might be expected that a rise of general systemic blood pressure would be detrimental to the subject because of the increased load which would thereby be placed upon the damaged ventricle. The risks of such an increase in arterial pressure (and of course in intraventricular pressure) are dependent upon the distribution of the damaged muscle and upon the extent of the damage. In other words, they depend on the ability of the remaining normal muscle in that region to withstand the muscle tensions generated. If it will not stand up to that pressure, then either the ventricular wall will rupture or an aneurysm will form. In a previous paper (Lowe, 1941) I have shown that either of these events will be determined largely by the particular muscle bundles involved, rather than by the total quantity of muscle tissue destroyed. Rupture of the ventricular wall or aneurysm formation is determined by the site of the infarct rather than by variations in the intraventricular pressure. A lowering of the pressures in the coronary circulation may cause an increase in the extent of the infarct and lead to rupture of the ventricular muscle, when a maintenance of the circulation pressure would have prevented such a development.

A study of the histological changes occurring in the infarcted muscle (Levine, 1929; Mallory *et alii*, 1939; Lowe, 1941) shows that it takes several weeks for a scar of dense fibrous tissue to form, but that if rupture or aneurysm formation is going to occur, it will commence when the ventricular wall is weakest—that is, during the early days of the lesion. Rupture usually occurs in the first week, occasionally in the second, and but rarely at later times (Mallory *et alii*, 1939).

The risk of raising the blood pressure after myocardial infarction is therefore mainly that of hastening the development of an aneurysm or of producing ventricular rupture. The raising of the blood pressure does not cause these events, for their occurrence is determined by the site and nature of the original infarct. Apart from these events, the raising of the coronary pressure can only do good.

It is obviously of more importance to maintain a good coronary circulation, with some added chance of hastening—not producing—cardiac rupture, than to keep the cardiac output minimal by absolute rest and so depress the coronary circulation and probably extend the area of the infarct. It is not suggested that general rest is not needed, but that absolute rest (Mallory *et alii*, 1939), by keeping blood pressure low, defeats its objective of preventing aneurysm formation or ventricular rupture. It also appears that in cases in which a gross fall in blood pressure has occurred, active steps to raise it to more normal levels are justified, because an improved coronary circulation may prevent an otherwise fatal outcome.

Conclusions.

It is possible to produce localized areas of necrosis in cardiac muscle, at predetermined sites and of controlled extent, by the injection of small amounts of concentrated phenol solutions.

These observations show that the destruction of localized areas of cardiac muscle, of the sizes indicated, produces no permanent disturbance of systemic blood pressure immediately after destruction, irrespective of the particular muscle bundle involved.

These experiments yield no clue to the causal factors of the fall of blood pressure observed, at least in later periods, by clinicians and experimental workers.

Summary.

1. A new method of producing localized myocardial death in dogs is described.

2. A series of observations on the immediate effect of localized myocardial death on the general systemic circulation is presented.

3. The effects on the myocardium of falling blood pressure after myocardial infarction are discussed, and the conclusions are applied in the treatment of human myocardial infarction.

Bibliography.

D. I. Abramson, C. Shookloff and N. M. Fenichal: "A Study of the Variations of the *RS-T* Segment in Experimental Ventricular Trauma", *American Heart Journal*, Volume XII, 1936, page 174.

D. W. Bennet and A. N. Drury: "Further Observations Relating to the Physiological Activity of Adenine Compounds", *The Journal of Physiology*, Volume LXXII, 1931, page 288.

A. E. Coates: "Later Results of Injection of Sympathetic Ganglia for Angina Pectoris", *Royal Melbourne Hospital Clinical Reports*, Volume XI, 1940, page 40.

L. Gross, G. Schauer and M. Mendlowitz: "Hæmodynamic Studies in Experimental Coronary Occlusion, Changes in Arterial Pressure", *American Heart Journal*, Volume XVI, 1938, page 278.

H. F. Haney, M. C. Borman and W. J. Meek: "The Relation Between the Position of Experimental Myocardial Lesions in the Dog and the Changes in the *RS-T* Segment of the Electrocardiegram", *The American Journal of Physiology*, Volume CVI, 1933, page 64.

C. H. Kellaway and R. Trethewie: "The Liberation of Adenyl Compounds from Perfused Organs by Cobra Venom", *The Australian Journal of Experimental Biology and Medical Science*, Volume XVIII, Part I, 1940, page 63.

B. Kisch, L. H. Nahum and H. E. Hoff: "The Predominance of Surface over Deep Cardiac Injury in Producing Changes in the Electrocardiogram", *American Heart Journal*, Volume XX, 1940, page 174.

S. A. Levine: "Coronary Thrombosis: Its Various Clinical Features", *Medicine*, Volume VIII, 1929, page 245.
T. E. Lowe: "Some Principles Governing the Supply of Blood to the Myocardium in Occusive Arterial Disease", *American Heart Journal*, Volume XXI, 1941, page 326; "Some Observations on Myocardial Infarction", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1941, page 693.
G. R. Mallory, Paul D. White and J. Salcedo-Solgar: "The Speed of Healing of Myocardial Infarction", *American Heart Journal*, Volume XVIII, 1939, page 647.
J. S. Robb, J. G. F. Hiss and R. C. Robb: "Localisation of Cardiac Infarcts", *American Heart Journal*, Volume X, 1935, page 287.
A. de Waart, C. J. Storm and A. K. J. Konmans: "Ligation of the Coronary Arteries in Javanese Monkeys", *American Heart Journal*, Volume XI, 1936, page 676; Volume XII, 1936, page 70; Volume XII, 1936, page 184.
C. J. Wiggers: "Physiology in Health and Disease", 1937, page 379.

THE INTERACTION BETWEEN ANTI-A AGGLUTININS IN GROUP B SERUM AND RECEPTOR SUBSTANCES IN GROUP A SERUM.

By LUCY M. BRYCE AND RACHEL JAKOBOWICZ,

From the Walter and Eliza Hall Institute, Royal Melbourne Hospital, and the Red Cross Blood Transfusion Service (Victoria Division).

IN a previous paper[1] we recorded variation in the interaction between the anti-A agglutinins in group B serum and the receptors in group A serum, apparently referable to individual differences in the former. This paper deals with further investigations on this point.

In 1938, Dahr[2] found that human anti-A serum contained at least two partial agglutinins, which he called x and y, and that there were corresponding fractions X and Y in the receptor-substances of group A red cells, and in the saliva of "A-excretors". He found, however, that the composition of the receptor substance in saliva could differ from that of the red cells in the same subject.

These observations of Dahr's followed those of Friedenreich and With,[3] who had recorded somewhat analogous findings in respect of the human B-agglutinogens and anti-B agglutinins; their work suggests the existence of at least three partial agglutinogens and agglutins which they call B_1, B_2 and B_3, and β_1, β_2 and β_3 respectively. They state that it is rare to find an anti-B serum containing all three partial agglutinins, and also that the composition of the B-receptor substances may be different in the cells and body fluids of the same subject.

We think that the variations we have encountered in serum may be explained by an hypothesis similar to that of Dahr; we postulate, however, the existence of three components in both receptor substance A and the anti-A agglutinins, which we refer to as X, Y and Z, and x, y and z respectively.

Technique.

Twenty-six specimens of B serum have been examined. For practical purposes these have been divided into five batches of five or six, and every B specimen in a batch has been tested with the same series of six to ten A specimens. Mixtures were made of 0·1 cubic centimetre of each B specimen with 0·1 cubic centimetre of each A specimen and with 0·1 cubic centimetre of saline solution as a control. These mixtures were allowed to stand for two hours at room temperature. They were then titrated in serial doubled dilutions with A cells (sub-group A_1) of known average sensitivity. Readings were taken after two hours at room temperature. The reduction of agglutinins effected by the mixtures of A and B serum, as compared with that obtained by the mixture of B serum with saline solution, was noted. If agglutinins in the serum mixtures were present in the same serial dilution as in the serum and saline solution mixture, the reduction is recorded as "–", if in one dilution lower, as "±"; if in more than one dilution, but less than two lower, as "+"; if in two or more lower, as "+ +".

The results obtained in Batches I and II, which are illustrative of all those examined (the others being omitted in the interests of economy of paper), are shown in Table I. In this table, the composition of the A and B specimens of serum in terms of the postulated components X, Y and Z, and x, y and z, is also indicated. Thus, for example, it will be seen that the titre of B specimen I was very little reduced by any A specimen except number 8. This could be explained on the assumption that B specimen I contained all three components x, y and z, and that it would therefore be completely or strongly reduced only by an A specimen containing all three receptor-substances X, Y and Z. Conversely, since A serum 8 strongly reduces every B specimen with which it is mixed, we assume that it must contain all three components X, Y and Z. Similarly the interactions of the other A and B serum mixtures can, we consider, be explained by the composition in terms of X, Y and Z and x, y and z, which are indicated in Table I.

It will be noted that four A specimens (numbers 9, 10, 15 and 16) are classified as sub-group A_2. This classification refers actually to the red cells of the blood samples from which these specimens were obtained. A distinguishing property of cells of sub-group A_2 is the absence

TABLE I.

Reduction of Anti-A Titre of B Serum after Absorption with A Serum.

A Serum.	B Serum.									
	1 (16).[1] $x+y+z$.	2 (16). $x+y$.	3 (16). $x+z$.	4 (16). x.	5 (128). x.	6 (32). $x+y$.	7 (32). $x+y+z$.	8 (16). x.	9 (16). x.	10 (16) x.
Batch I.										
1. A_1 X ..	±	++	+	+	+					
2. A_1 Y ..	–	+	–	–	–					
3. A_1 Y ..	–	++	–	–	–					
4. A_1 $X+Y$	±	++	±	+	+					
5. A_1 $X+Y$	±	++	±	+	+					
6. A_1 $X+Y$	±	++	±	+	+					
7. A_1 $X+Z$ ..	±	±	+	+	+					
8. A_1 $X+Y+Z$	++	++	++	++	++					
9. A_2	–	–	–	–	–					
10. A_2	–	–	–	–	–					
Batch II.										
11. A_1 X ..						±	±	+	+	+
12. A_1 X ..						±	±	++	+	++
13. A_1 X ..						±	±	++	++	++
14. A_1 X ..						–	–	+	+	+
15. A_2 X ..						–	–	+	+	+
16. A_2						–	–	–	–	–

[1] The figures in parentheses indicate the anti-A titre after two hours' contact with an equal volume of saline solution.

or relative lack of avidity for anti-A agglutinins. In the majority of cases (as in numbers 9, 10 and 16) a similar property is found in the serum of such blood. Dahr,[1] however, noted that the saliva of a subject whose red cells belonged to sub-group A_2, could belong to sub-group A_1; a similar phenomenon appears in the case of our A serum 15, which reacts as an A_1 serum containing x receptor substances, although the cells of this blood belonged to sub-group A_2.

The great majority of the specimens examined have given clear-cut results such as those illustrated, which can be explained on the hypothesis of the existence of at least three qualitatively different partial receptor substances and agglutinins. Occasional exceptions have, however, been encountered. In two instances serum from blood with cells of sub-group A_1 failed to reduce any of the B specimens with which it was mixed. The subjects from whom the serum came are probably to be regarded as examples of the "non-excretors" described by Schiff.[2]

In a few other cases the intensity of the interactions varied in a way which suggested that the components X, Y and Z and x, y and z might differ quantitatively as well as qualitatively.

The avidity of the red cells, the serum and the urine may vary in the same subject. This variation, and also a comparison between the sub-groups A_1 and A_2, is shown in the following experiment:

B specimens 6, 7 and 8 (see Table I) were absorbed with the red cells, serum and urine of two additional group A subjects, one belonging to sub-group A_1 and the other to sub-group A_2 (not included in the above series A_1—16), the technique used being similar to that described above. The results obtained are shown in Table II.

TABLE II.
Reduction of Anti-A Titre of B Serum after Absorption with A Serum, Urine and Cells.

Testing Substance.	B Serum.		
	6 *x-y-z.*	7 *x-y-z.*	8 *z.*
Serum K (A_1) X	±	±	++
Serum L (A_2)	−	−	−
Urine K (A_1) X	−	−	±
Urine L (A_2)	−	−	−
Red cells K (A_1)	+	++	++
Red cells L (A_2)	−	−	+

These results illustrate the qualitatively unselective and powerful reducing power or avidity of the A_1 red cells as compared with that of the serum and urine of the same subject, and also the difference between the avidity of A_1 and A_2 receptor substances. Even in respect of the latter, however, the red cells showed some reducing power, which was absent from the serum or urine.

Discussion and Summary.

We have made no attempt to separate the three components postulated for A agglutinogens and anti-A agglutinins. The problem was approached primarily from the practical standpoint, as a result of the unexpected absence of reduction of titre which occurred in a mixture of specimens of A and B serum previously reported.[3] The further investigations described in this paper indicate that there is a considerable qualitative and possibly also quantitative variation in the components of the A antigen and the anti-A agglutinin. It is not possible, therefore, to predict beforehand how much reduction any one specimen of A serum will effect in the anti-A titre of any specimen of B serum. For this reason, it is important to mix as many samples of A and B serum as possible, if constant and effective reduction of agglutinin titre is aimed at, when preparing pooled human serum for therapeutic use.

References.

(1) R. Jakobowicz and L. Bryce: "The Iso-Agglutinin Titre of Pooled Serum or Plasma", THE MEDICAL JOURNAL OF AUSTRALIA, March 15, 1941, page 318.

(2) P. Dahr: *"Ueber die Zusammensetzung des menschlichen Anti-A Agglutinins in verschiedenen Anti-A Seren und deren Bedeutung für den Nachweis von A in Secreten"*, *Zeitschrift für Immunitätsforschung und experimentelle Therapie*, Volume XCIII, 1938, page 480.

(3) V. Friedenreich and S. With: *"Ueber B-Antigen und B Antikörper bei Menschen und Tieren"*, *Zeitschrift für Immunitätsforschung und experimentelle Therapie*, Volume LXXVIII, 1933, page 152.

(4) F. Schiff and H. Sasaki: *"Ueber die Vererbung des serologischen Ausscheidungstypus"*, *Zeitschrift für Immunitätsforschung und experimentelle Therapie*, Volume LXXVII, 1932, page 129.

INVESTIGATION OF FUNGOUS INFECTION OF THE FOOT IN A MILITARY CAMP.

By L. W. LINN, M.B., B.S.,
Honorary Dermatologist, Adelaide Children's Hospital,

AND

F. R. MAGAREY, M.D.,
Adelaide.

FUNGOUS infection of the foot was reported to be widespread amongst men in military camps in South Australia. A large variety of therapeutic measures, both prophylactic and curative, was being carried out in the absence of definite ideas as to their effectiveness. As a result of this, Colonel A. R. Southwood, D.D.M.S., Fourth Military District, arranged for an investigation to be made into the nature, degree and incidence of foot infection in soldiers. This was carried out at the military camp, Woodside, from April 23 to May 8, 1941.

Six hundred and fifty men were examined, and of these 402 were found to have some abnormality between the toes. The men came from various units under training during this period.

Method of Investigation.

The regimental medical officer of the unit held a foot parade and selected men with any lesion between the toes. The same day these men were examined by the dermatologist, who took scrapings for direct microscopic examination and for incubation, at the same time classifying the clinical condition present. The scraping for microscopic examination was cleared in 30% aqueous solution of potassium hydroxide, sealed with "Vaseline" and examined after eight hours. The structures were found not to change between that time and seven days. Each specimen was examined independently by two workers, either the pathologist or dermatologist and an army staff nurse specially trained in microscopic work.

The specimen for incubation was inoculated directly onto a Sabouraud's slope in a McCartney bottle and incubated at 22° C. The media were examined every second day, and if no mould or fungus had appeared after ten days they were discarded.

For identification of the fungus the method pursued was reinoculation from a colony onto a square centimetre of Sabouraud's agar between a sterile microscopic slide and a cover slip, and incubation in a sterile Petri dish at 25° C. The mycelium, hyphæ and spores could be examined microscopically without disturbance of the specimen.

Results of Investigations.

Clinical.

The lesions examined were classified as follows: scaling, 256; macerated skin, 136; fissure with scaling, 4; fissure with macerated skin, 6. Of the men investigated, none was found to be suffering from any disability sufficiently severe to interfere with his training, and in no case was the condition found to have spread onto the dorsum or plantar surface of the foot.

Of the 402 cases examined, the provisional diagnosis of tinea was made in 257. The others were classified as either "suspect" (90) or "not tinea" (55).

Microscopic.

A positive diagnosis could be made in only seven cases; in six of these the clinical diagnosis was tinea, and the other was a suspected case. The percentage of fungous infections diagnosed microscopically in cases associated

with any abnormality between the toes was 1·7; in those in which the diagnosis of tinea or of suspected tinea was made, it was 2·0.

In the seven cases microscopically yielding positive findings, the lesions between the toes were as follows: scaling, 5; macerated skin, 2.

Cultural.

Epidermophyton was isolated and subcultured in four cases. The abnormality between the toes and clinical diagnosis in these cases were as follows: (i) macerated skin, tinea; (ii) macerated skin, suspected tinea; (iii) macerated skin, not tinea; (iv) scaling, suspect.

Epidermophyton was not grown in any case in which direct microscopic examination gave positive findings.

Discussion.

The striking feature of the investigation was the discrepancy between the clinical and the laboratory diagnosis, and the lack of correlation between the direct microscopic and the cultural findings. It was to be expected that the fungus, if present, should have been detected more frequently by the cultural than by the microscopic method; but as the reverse was the case, this can possibly be explained by the fact that the fungus had died, perhaps as a result of treatment, and had left definite traces of mycelium in the desquamated skin. In the four cases in which *Epidermophyton* was recovered, and in which no direct microscopic evidence of fungous infection was found, the fungus must have had a saprophytic existence.

The ætiology of the majority of the lesions is probably influenced by several factors. In civil life most men are unaccustomed to much walking, and shoes and cotton socks are generally worn; whereas in camp, men are obliged to do a considerable amount of marching, and wear boots and woollen socks. As a result the feet become overheated and sweat excessively. The hyperhidrosis causes maceration of the skin, which, when dry, desquamates. Infection thrives better on dead than on living tissue, and it is obvious that persistent maceration results in the death of the horny layer of the skin. In view of the low incidence of fungus found in this investigation, it is suggested that one ætiological factor may be a low-grade bacterial infection, which would thrive in the ideal medium of warm, moist, dead tissue. It was noted that men with well-spaced toes nearly always had healthy skin.

It is also possible that chemical irritation may play some part in damaging or destroying the epidermis. When socks are washed under camp conditions the soap is often inadequately rinsed from them, and the alkali from coarse soaps would certainly act as an irritant when in constant contact with the skin.

Treatment.

In spite of the mildness of the condition at the time of the investigation, it is suggested that many of those men with lesions might suffer an aggravation of the condition from the rigours of active service, particularly in hot weather. On this assumption, it is recommended that all men should receive instruction in foot hygiene and simple prophylactic treatment.

The following routine is suggested: (i) Weekly foot inspection by the regimental medical officer. (ii) Thorough drying of the feet after washing. (iii) Removal of the socks when possible and exposure of the feet to the air; the socks also to be changed as often as possible, washed and rinsed well to free them from soap. (iv) A daily swabbing of the feet with methylated spirits, followed by dusting with a powder containing 3% salycilic acid, 25% *Pulvis Amyli* and *Pulvis Acidum Boricum* and talcum of each to 100%, this operation to be supervised by the hut or tent non-commissioned officer. (v) Daily scrubbing and disinfecting of shower boards, which should be placed in the sun to dry for as long as possible.

Conclusions.

1. Fungous infection of the foot is far less common than is generally supposed.

2. The fact that the lesions can be present without infection by the fungus and that the fungus can be present without invading the skin is presumptive evidence that the fungus is not necessarily the only causal agent of the disease. It can also exist either as a saprophyte or as a secondary invader.

3. Clinically it is found impossible to separate those lesions caused by fungus from those due to other factors.

4. It is suggested that the ætiological factor of the majority of the lesions is either a chemical irritant or a low-grade bacterial infection frequently associated with and aggravated by hyperhidrosis.

Acknowledgements.

We wish to express our thanks to Colonel A. R. Southwood, Deputy Director of Medical Services, Fourth Military District, for permission to publish the results of this investigation, and to acknowledge the valuable advice given to us by Professor Platt, Professor of Bacteriology, University of Adelaide, concerning the laboratory technique.

Reports of Cases.

A CASE OF FIBROMA OF THE EXTERNAL AUDITORY MEATUS.[1]

By HERBERT EARNSHAW,
Brisbane.

THE external auditory meatus is not a common site for the development of fibromata. The occurrence of such a tumour in this site has not been previously recorded, though Moriconi,[1] in 1937, described a case of fibroma of the auricle, and de Souza Neves,[2] in 1938, a case of massive fibroma beginning in the "*pavillon*", which is presumably the concha of the ear.

Clinical Record.

The details of this case are as follows. Mr. D. was referred to me by Dr. L. P. Musgrave, of Redcliffe, in November, 1939. He complained of a growth in the right ear. A lump had first been noticed seventeen years previously. No treatment was sought for two years, when the patient consulted a doctor. Mr. D. was not informed of the diagnosis, and he was given ointments to use. About thirteen years ago an attempt was made to excise the lump, but profuse bleeding occurred and the operation was abandoned; the hæmorrhage was controlled with difficulty by packing. About a year before my seeing the patient the swelling had been incised, and again profuse hæmorrhage occurred, controlled with difficulty. The previous history and family history were irrelevant.

On examination the left ear presented a tense swelling measuring one inch by half an inch by half an inch, which projected from the meatus. The surface of the swelling was irregular and rather "scrotal" in appearance and showed one scar. It was non-fluctuant, but somewhat springy to palpation; the swelling was translucent. The tumour completely filled the external auditory canal and projected almost to the antitragus. The base of the swelling was situated in the posterior wall of the external auditory canal; the distal border of the base was at the concho-meatal junction. It was not possible to determine the mesial limit. The superior and inferior limits of the tumour were approximately the middle lines of the superior and inferior walls of the meatus respectively. Figure I gives a good idea of the appearance. The part projecting onto the concha was movable. The external auditory meatus exuded a thin, watery discharge containing epithelial débris. The tympanic membrane was not visible.

The provisional diagnosis of sebaceous cyst of the external auditory meatus was made; a large bore needle was passed into the centre of the tumour and an attempt was made to perform aspiration through this. Aspiration produced nothing, but on the removal of the needle a spurt of blood followed, which pulsated strongly, and the blood was ejected for about two inches till controlled by pressure.

At operation on November 6, 1939, a posterior flap was cut with the base towards the occiput and the apex extending

[1] The patient was shown at a combined meeting of the Queensland Branch of the British Medical Association and the Brisbane Hospital Clinical Society on August 1, 1941.

onto the posterior surface of the pinna; the cartilage of the pinna was exposed, and the soft part of the meatus was freed from the bony meatus for about three-quarters of an inch. The tumour was then excised *in toto* with the cartilage subjacent to it. Hæmorrhage was free, but was controlled by pressure. The flap was then replaced in position and sutured with many interrupted vertical mattress horsehair sutures.

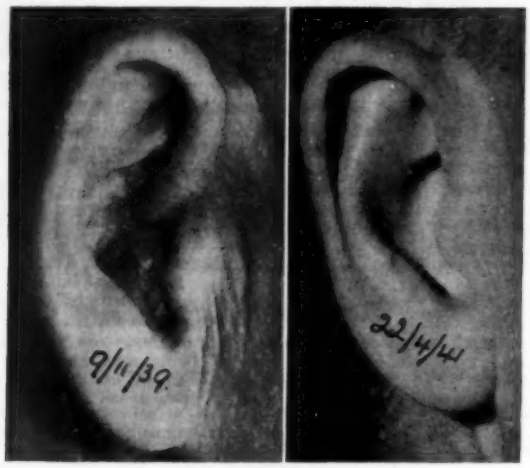

Figure I.

The external auditory meatus was now cleansed by mopping with swabs moistened with hydrogen peroxide solution. The tympanic membrane was found to be intact. This was covered with a small gauze tampon, and a mould of Kerr's composition was made to fit the meatus. A thin razor graft taken from the arm, trimmed to the necessary size and shape to cover the defect, was applied on the Kerr's mould to the denuded area, the auricle was packed with dry gauze and covered with a cotton wool pad, and a tight bandage was applied.

The ear was left untouched for four days, when the outer dressing was changed carefully so as not to disturb the mould, and the sutures of the posterior flap were removed. The mould was left undisturbed for twenty days, when it was removed and replaced by ribbon gauze moistened with liquid paraffin. The tampon over the tympanic membrane also was removed at this time. The paraffined gauze was dispensed with after another six days, when the deficient area was found to be covered with healthy epithelium.

On examination fourteen days later it was found hard to distinguish the edges of the incision in the meatus. The specimen was submitted to Dr. Noel Gutteridge for microscopic examination, and his report was as follows: "Section shows a fibroma, covered by normal squamous epithelium, and with a cartilaginous base. No evidence of malignancy seen."

The patient was again seen on April 22, 1941, when the second photograph was taken. The auricle and meatus were quite normal and there were no signs of recurrence. No meatal stenosis was present.

Summary.

A case of fibroma of the external auditory meatus treated by surgical excision and skin grafting is presented.

References.

(1) L. Moriconi: *"Fibroma voluminoso del padiglione auricolare"*, *Gazzetta internazionale di medicina e chirurgia*, Volume XLVII, May 15, 1937, page 286.
(2) A. C. de Souza Neves: *"Un volumineux fibrome du pavillon auriculaire"*, *Paris médical*, Volume II, October 8, 1938, page 258.

Reviews.

AN IMPORTANT AUTOBIOGRAPHY.

If, as Emerson says, great men are to be distinguished by the range of their activities, then Hugh Hampton Young is surely worthy of a prominent place among the leaders of contemporary medicine. Now in his seventy-first year and an acknowledged authority in urological science, Young has in "A Surgeon's Autobiography" written the story of his extraordinarily varied and eventful life.[1] The book is certain to be warmly welcomed, for the author has presented a vast amount of material in a light and entertaining fashion that should appeal to both the medical and the general reader. Yet this work is something more than an outline of the career of a great surgeon. It tells a fascinating story of the growth of the science of urology during the last half-century and the great contributions made by Hugh Young and his associates to the advancement of surgery of the genito-urinary tract. But the author has not restricted his writing to matters of purely medical interest; his observations and reminiscences range over a wide field.

Of Confederate stock, Hugh Hampton Young was born at St. Antonio in Texas, and the opening chapters deal with his boyhood days, his early schooling and undergraduate activities in the University of Virginia. Graduating in arts and medicine in the short period of four years, Young was for a time engaged in private practice before entering upon a course of surgical training at Johns Hopkins Hospital, Baltimore. His choice of school was fortunate. Although established only five years, Johns Hopkins under the direction of Osler, Welch and Halstead had attained first place among American medical schools. In such an environment it was inevitable that Hugh Young, a born scientist, should soon attract the attention of his superiors. His remarkable researches into the bacteriology of genito-urinary infections and his improvements in methods for their surgical treatment were rewarded by an appointment after only three years' residence as director of the hospital urological department, and Young forsook general surgery for the specialty the advancement of which was to become his life's work. Of these days at Johns Hopkins and of the men with whom he worked the author writes with gratitude and affection as well as a good deal of humour.

In a section of some hundred pages dealing with the development of his specialty Young then gives a detailed description of the various pathological conditions encountered in genito-urinary practice and the operative procedures he has devised for their control or cure. This chapter is written in non-technical language and is superbly illustrated; it should be of most value to the general reader, for whom it is written.

A notable event in the author's career was the opening in 1914 of the James Buchanan Urological Institute. Endowed by the colourful "Diamond Jim" Brady, the institute has under Young's direction made great progress in research and undergraduate teaching in genito-urinary surgery. Another noteworthy achievement was the publication in 1917 of the first number of *The Journal of Urology* under the editorship of Hugh Young. In spite of difficulties created by the war the journal continued to appear until in 1920 it became the official organ of the American Urological Association, controlled by an editorial board with Young editor-in-chief. Since its establishment *The Journal of Urology* has achieved an important place in medical literature and is perhaps the leading journal of its kind.

Upon the entry of the United States of America into the Great War, Hugh Young immediately offered his services to the Surgeon-General and on his own suggestion was sent to Europe to study methods for the control of venereal disease adopted by the British and French armies. His observations upon this most important and difficult subject could be studied with profit by all concerned with army medicine. The remarkably low incidence of venereal disease among troops of the American Expeditionary Force is evidence of the thoroughness with which the problem was met. As Director of Urology to the army Young was in close touch with the United States troops in France and gives an interesting account of their disposition and the actions in which they were engaged. Some good stories are also told of days in London with President Wilson's retinue following the armistice.

The closing chapters of the work deal mainly with the author's extraprofessional activities, his labours in the public service, his constant endeavours to further health legislation in the State of Maryland and to enrich the cultural life of his beloved Baltimore. His world travels, his musical and aviation interests, his adventures with rod and gun, and his stock of excellent stories all reveal Hugh Young's varied tastes and diverse interests. His book records the life of an outstanding surgeon; his readers will lay it down thankful for a glimpse of the soul of a great American.

[1] "A Surgeon's Autobiography", by H. Young; 1940. New York: Harcourt, Brace and Company. Medium 8vo, pp. 566, with over 100 drawings and 3 colour plates. Price: 30s. net.

The Medical Journal of Australia

SATURDAY, OCTOBER 18, 1941.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

INDUSTRIAL FATIGUE AND AUSTRALIA'S WAR EFFORT.

So much is heard of Australia's "war effort" that many people take it for granted, first of all, that a strong and continuous effort to win the war is being made, and, secondly, that the effort is so intelligently directed that neither physical nor mental energy is being wasted. But scepticism is healthy in matters political as well as scientific, and incidentally would appear to be the more necessary in view of the displays of party pyrotechnics that have been staged from time to time at Canberra. Every person in the Commonwealth who has expert knowledge that can be of use to the nation in its present need has a right to demand that it should be used to the best advantage. The medical profession of Australia is not satisfied that the Commonwealth Government is making full use in the field of industry of knowledge that is reliable, proven and capable of application. For this reason we welcome the paper by Dr. H. M. L. Murray published in this issue, and declare it to be one of the most important that has appeared in this journal since the outbreak of war.

In April of this year fatigue in relation to war-time industry was discussed in these pages. This was shortly after the Federal Council of the British Medical Association in Australia at its meeting in Melbourne, following a discussion on the hours of work for Air Force trainees, decided to refer to the Prime Minister the whole question of industry and fatigue. In our April discussion it was pointed out that man is not a machine and that his efficiency in industry depends on many factors, but chiefly on his muscular and nervous systems. It was also stated that though this was learned somewhat painfully in the war of 1914-1918, in the present war the authorities in England have had to be reminded over and over again of the facts established in the earlier conflict. Reference was also made to Emergency Report Number 1, entitled "Industrial Health in War", and issued in Great Britain by the Industrial Health Research Board; special emphasis was laid on the following statement from its preface: "The need at the moment is rather for application of knowledge previously gained than for new researches, although fresh problems for investigation may present themselves as the war proceeds." The Federal Council, as determined at its March meeting, wrote to the Prime Minister. At the meeting of the council held at Sydney in September the General Secretary reported that he had received a reply, dated April 17, 1941, from Mr. T. J. Collins, who wrote on behalf of the Acting Prime Minister. In this reply it was stated that there was under existing conditions no alternative to the working of twelve-hour shifts in munition factories. The letter continued as follows:

> It will be appreciated that the necessity of the moment is production to the utmost extent possible, and that can be obtained only by working the plant and equipment for twenty-four hours a day for as many days as it is possible consistent with the upkeep and maintenance of the plant. In ordinary circumstances the ideal arrangement would be to work three shifts; but owing to the very serious shortage of skilled supervisors, of tradesmen for setting up the machines used by unskilled operators and of tool and gauge makers necessary to produce manufacturing equipment used by unskilled operators, it is impracticable to do any more at present than impose working hours of two long shifts.

The members of the Federal Council felt that this letter was based on views incompatible with a proper understanding of fatigue and its effects, and they were so convinced that a persistence in the policy dictated by it would greatly lessen output of munitions and other war material that they determined not to let the matter rest, but to approach the Prime Minister once more.

Dr. Murray in his article traverses most of the ground covered in our discussion last April, but he goes further. He deals in a convincing fashion with the oft-repeated and false statement that the machine sets the pace. We do not intend to follow his argument; readers must study his paper for themselves. His reference to completely automatic machines is important, and it is well to remember that in Australia "one does not often see a factory in which operatives have to work to capacity to look after their quota of automatic machines". Dr. Murray's mention of German conditions is also interesting. Those who wish to pursue this subject further will find many details of German conditions described by P. Waelbroeck and I. Bessling in the League of Nations publication, the *International Labour Review*, for February, 1941. In the course of their article, which is entitled "Some Aspects of German Social Policy under the National-Socialist Régime", these authors point out that in 1936 the ten-hour day was largely substituted for the eight-hour day, especially in the metal-working, building and similar industries. In May, 1938, Marshal Göring declared: "He who wishes to work for the recontruction of the Reich must not only work eight hours a day, but must work as he has never done before." In 1938 it was found necessary to make the regulations more flexible. When hostilities broke out in September, 1939, all restrictions on normal hours of work for adult men were removed. Holidays of all kinds were abolished, and the authorities were empowered to suspend all legislative provisions prohibiting Sunday work, night work *et cetera*. In less than three months "it was found neces-

sary to restore the regulations in a certain measure". The President of the National Employment Office observed: "Attention must however be paid to the physical and mental limits to any extension of hours. Any abuses lightly committed in this respect will have to be paid for dearly sooner or later." The Director of the Ministry of Labour said: "It must be recognized that it is a national necessity to avoid, despite all the indispensable efforts, any excessive fatigue that can be avoided, so as to maintain intact a strong and healthy nation fit to defend itself." Waelbroeck and Bessling state that in many respects the tendencies which find expression in such measures of restoration of restriction of working hours are not peculiar to Germany today; "they are inherent in every war economy and can be found, at least in nucleus, in most countries where war or the risk of war has forced the sovereign interests of national defence to the foreground". Dr. Murray is more than justified in his conclusion that there is ample information on this subject available for all who have the intelligence to use it. We think that his words in this sentence have been particularly well chosen.

Australians, and particularly Australian manual workers, have the right to know why the powers-that-be in this country, in Dr. Murray's words, "care for none of these things". Why is there a "fatal fascination about multiples of the figure 'four'"? This fascination is evident even in the letter written for the Acting Prime Minister to the Federal Council, portion of which has been quoted. No one can possibly deny that the available evidence on this subject is sufficient to justify the drawing of conclusions; no one can deny that the evidence is reliable; no one can deny that according to this evidence it would be better for machines to be idle for four hours a day and for working hours to consist of ten-hour shifts. (Under some conditions eight hours might be more productive than ten.) Dr. Murray states that it seems quite impossible to "push into the comprehension" of the average production manager certain ideas. Why bother about the average production manager? It is into the comprehension of the Prime Minister, the Minister in charge of the production of munitions and his leading henchman that ideas have to be pushed. These men have to be made to see that they are fathering what Dr. Murray so well calls gross inefficiency, when in their dealings with workers willing to be led, they could produce a really magnificent war effort. When the Federal Council approaches the Prime Minister again, it should see that Dr. Murray's most valuable and courageous contribution is brought to his notice.

Current Comment.

NEW IDEAS ABOUT DIPHTHERIA ANTITOXINS.

WHEN medical men discuss their fatal cases of diphtheria the opinion is often expressed that the disease is not responding as well to treatment with antitoxin today as it did. There is no doubt that much larger doses of antitoxin than formerly (in terms of "units") are now generally administered to very sick patients and yet numbers of fatalities occur, sometimes in a disquietingly quick succession, which is ascribed with some truth to the prevalence of a very virulent strain of the diphtheria microbe at the particular time. But, as R. A. Q. O'Meara[1] has recently reminded the profession, the diphtheria which was treated with such brilliant results in 1894 by Ehrlich, Kossel and Wassermann, Roux, Martin and Chaillou, was of a very severe type, causing much intoxication and a considerable mortality rate; yet they used quite small doses of antitoxin. There can be no reasonable doubt, then, that some antitoxic substance which used to be present in diphtheria antitoxins is now less abundant or lacking. It would appear that the adoption of the Ehrlich "unit"—based on the amount of antitoxin required to save the life of a guinea-pig imperilled by a lethal dose of toxin—as the measure of the theoretical efficacy of diphtheria antitoxin, was responsible for this. Manufacturers have refined and concentrated their products; they have largely eliminated bodies producing anaphylactic reactions to be sure, but chiefly they have striven to increase the number of units in a given volume. What if the Ehrlich unit is not a true measure of therapeutic potency? O'Meara asserts that the modern refinements of diphtheria antitoxin have never received any but the most superficial clinical tests for their influence on therapeutic value.

The manner in which an antitoxin "neutralizes" a toxin is as yet only vaguely understood. If to a unit of antitoxin a unit of toxin be added, a neutral mixture is obtained; but if to a unit of antitoxin a fraction of a unit of toxin be added and the mixture be allowed to stand for a time, the antitoxin is no longer capable of neutralizing the remainder of a unit of toxin supplied in a second addition; this is known as the Danysz phenomenon, and was shown to apply to diphtheria toxin and antitoxin in 1904. The antitoxin is said to be rendered non-avid by the first addition; in this non-avid state it is no longer capable of holding toxin firmly and on dilution dissociates from it. As long ago as 1913 a medical controversy raged round the question of whether antitoxin owed its relative efficiency to its "unitage" or to its relative "avidity".

Now, O'Meara claims to have shown that diphtheria toxin contains two toxic bodies. One of these ("substance A") is highly lethal for the guinea-pig, but little of it is produced in the body in human diphtheria, although "it has hitherto been assumed that hypertoxic diphtheria is caused by its production in excessive amounts". Conversely, laboratory toxin contains relatively little of the other toxic body ("substance B"); but it is this, according to O'Meara, which determines the occurrence of hypertoxic diphtheria. Corresponding to the two toxic bodies in toxin there are two antitoxic bodies in antitoxin. The antitoxins in common use today contain a high proportion of the antibody to substance A, but are deficient in the antibody to substance B. When these antitoxins are used in the treatment of hypertoxic diphtheria, O'Meara concludes, substance B is not neutralized and the antitoxin is rendered non-avid. It therefore dissociates from its combination with toxin when it is diluted in the blood stream and the intoxication continues unabated.

C. J. McSweeney,[1] the medical superintendent of a Dublin fever hospital, with long experience, has administered to sixteen patients suffering from hypertoxic diphtheria of exceptional severity an "avid" antitoxin known to be rich in the antibody to O'Meara's "substance B", and he states that never before had he seen such satisfactory results in this type of diphtheria. Unfortunately, this particular type of antitoxin, avid and rich in the antibody to substance B, cannot yet be produced at will, and the batch used by McSweeney appears to have been a freak of Nature. It is interesting to recall in this connexion the statement recently made by D. C. R. Vickery in this journal that a successful vaccine for the treatment and prophylaxis of whooping cough was produced in this country a dozen years ago by the accidental use of organisms in a certain phase of growth; subsequent batches of vaccine prepared by the same methods by the same makers failed to achieve the same success, and the explanation has only recently been forthcoming in the discovery that the phase of growth of the organisms used greatly influences the efficacy of the vaccine.

[1] *The Lancet*, February 15, 1941.

[1] *The Lancet*, February 15, 1941.

No doubt O'Meara's concepts will have to run the gauntlet of close criticism, barracked (we may well imagine) by the shades of the protagonists of many half-forgotten controversies of the early part of the century. But it will undoubtedly stimulate research. Recently R. A. Kekwick and other workers at the Lister Institute of Preventive Medicine have described electrophoretic analyses of the composition of diphtheria antitoxic serum, and have shown that of three separable globulin components, two, the β and γ fractions, have antitoxic activity, although there are striking differences between them.[1] The relative and absolute amounts of the β and γ antitoxins in the serum altered during the course of immunization of horses. The γ globulin (containing the γ antitoxin) tends to be precipitated in the salting-out method of serum concentration; also the γ antitoxin tends to be lost in the "pepsin process" of serum concentration.

EWING'S TUMOUR.

Since Ewing's original conception of a new type of bone tumour, "endothelial myeloma of bone", in 1921, this type of tumour has been the subject of numerous discussions. Especially has the question of its histogenesis been widely debated. Ewing's suggestion that the tumour originated from the vascular endothelium of the bone has never been uniformly accepted. A reticulo-endothelial derivation has been advanced by some authors, and others, especially the French school, have placed the origin in a primitive mesenchymal cell, multipotent and capable of differentiation towards endothelium or reticulo-endothelium or into blood-forming elements. The possibility has been ventilated that the lymphoblast or the hæmocytoblast may be the cell in which the tumour originates. Furthermore, it has been said that this tumour is not endothelioma, reticulo-endothelial sarcoma or myeloma, but that it is a round-cell sarcoma springing from undifferentiated embryonic mesenchymal cells situated in connective tissue about blood vessels in the Haversian canals. All these authors, however, at least accept the existence of a disease commonly called "Ewing's tumour of bone". This attitude is also reflected in a recent publication by H. W. Meyerding and J. E. Valls[2] in a review of primary malignant tumours of bone observed at the Mayo Clinic. But even the existence of Ewing's tumour as a separate entity has been questioned, in 1933 by Colville and Willis, later on by C. Sternberg, and recently again by Willis.[3]

Willis was able to show that on the only two occasions on which a typical Ewing's syndrome came under his observation it was produced by the metastasis of a neuroblastoma. On account of these findings and a review of the literature he comes to the conclusion that the occurrence of a primary growth of bone of this nature is still unproved. Willis's challenge has been taken up in a paper by F. W. Foote and H. R. Henderson[4] and in another paper by V. V. Gharpure.[5] The first authors report the case of a boy, fifteen years of age, in which a growth involving the right seventh, eighth and ninth ribs as well as the adjacent pleura and lung tissue was found. A portion of the seventh rib was surgically removed and microscopically examined. The diagnosis of Ewing's endothelial myeloma of bone was made. Foote and Henderson assert that they have been able to establish with certainty the origin of this type of tumour from vascular endothelium by showing vasoformative properties of the tumour cells in close proximity to tumour areas of quite diffuse arrangement. They are convinced that structures as seen by them could scarcely be duplicated by multiple myeloma, lymphosarcoma, metastatic neuroblastoma or metastatic bronchogenic carcinoma. The paper mainly contains a discussion of the histogenesis of these tumours, but does not give any information as to how the possibility of a primary tumour elsewhere has been excluded. No follow-up history is given, and Willis's criticism that only cases with a complete post-mortem examination can provide proof for the existence of an entity "Ewing's tumour" will still hold.

Gharpure in his paper forestalled much of this criticism by giving a description of a complete post-mortem examination in a similar case. A boy, fourteen years of age, had sustained an injury while playing two months prior to death. Immediately thereafter a painful swelling of the thigh was noted. Continuous fever developed and the tumour of the thigh increased in size until it reached by external measurement 76 centimetres in circumference, 43 centimetres in length, and 23 centimetres in thickness. At post-mortem examination the femur showed two pathological fractures in the middle of a very large cystic tumour. Save for the fact that the tumour surrounded the femur and had caused double pathological fractures, it would not have been possible to state whether it arose in bone or in adjacent soft tissues. No other tumour was found anywhere in the body, apart from a small nodule, one centimetre in diameter, on the external surface of the base of the right lung. The histological examination revealed an unusual structure in that very large numbers of pseudo-rosettes were present. It seems that in this case the possibility that the tumour was a metastatic growth can be excluded; but the case may be open to criticism from other points of view. Gharpure himself stresses some unusual features, such as the short clinical duration of only two months and the unusual macroscopic and microscopic findings, and it remains doubtful whether such an atypical case is a sound basis for general conclusions.

Gharpure mentions as further support for the existence of Ewing's tumour as an entity the record of cured cases in the collection of the Registry of Bone Sarcomas of the American College of Surgeons; but he discloses that in a recent review of these records by Stewart only eleven cases were accepted as examples of the disease. If only such a small number have withstood rigid criticism, the contention of Willis that the subject of Ewing's tumour is in a chaotic state seems to be justified and it will need much more conclusive evidence, especially in the form of complete post-mortem examinations in typical cases, before Ewing's tumour can be regarded as a well-established entity.

ELECTROCARDIOGRAPHY AFTER PNEUMONECTOMY.

In 1935 McGinn and White showed that electrocardiographic changes after massive pulmonary embolism might simulate those of posterior cardiac infarction. Later, Wood, who carefully studied tracings from chest leads, declared that the appearances were similar to those produced by acute *cor pulmonale* resulting from other disorders. It is thus possible to distinguish pulmonary embolism from coronary artery occlusion, the condition with which it is most likely to be confused clinically. Cyril G. Barnes states that in examining eleven patients who were undoubtedly suffering from pulmonary embolism he found only three whose electrocardiographic tracings resembled those of acute *cor pulmonale*.[1] He concluded that the emboli had not been large enough, and he sought a means of correlating electrocardiographic changes with the size of the vessel occluded. He therefore undertook an electrocardiographic study of patients before and after they had been subjected to lobectomy or pneumonectomy. Electrocardiographs from five patients subjected to lobectomy were similar to those taken before operation. A tracing obtained from one of six patients who had been subjected to pneumonectomy was abnormal; but in this case no tracing had been obtained prior to operation. No clinical evidence of failure of the right side of the heart was noted. Barnes concluded that if the right ventricular failure in pulmonary embolism was due to mechanical obstruction, "then more than 50% of the pulmonary artery must be occluded to cause it, in the presence of a healthy myocardium".

[1] *The Lancet*, May 3, 1941.
[2] *The Journal of the American Medical Association*, July 26, 1941.
[3] *The American Journal of Pathology*, May, 1940.
[4] *Ibidem*, July, 1941.
[5] *Ibidem*, July, 1941.

[1] *Proceedings of the Royal Society of Medicine*, July, 1941.

Abstracts from Medical Literature.

PATHOLOGY.

The Occurrence of Mitotic Divisions in Glomeruli in Glomerulonephritis and Malignant Sclerosis.

P. H. HARTZ, ARY VAN DER SAR AND AL VAN MEETEREN (*The American Journal of Pathology*, July, 1941) attempt to prove that, contrary to current opinion, mitotic divisions in the glomeruli in glomerulonephritis and malignant sclerosis are not exceptional. It is generally agreed that the essential lesion in glomerulitis is an increase in the number and size of the endothelial cells. On the other hand, most authors agree on the absence of mitotic divisions in the endothelial cells; neither are mitoses in the epithelial cells mentioned in recent publications. It was therefore concluded that if cell division actually occurred, it was largely of the amitotic type. As experienced cytologists either consider amitosis very rare in mammals or doubt the existence of real amitosis, this is not a satisfactory solution of the problem. Other authors state simply that there is an increase in the number of the endothelial cells without telling how this increase is brought about, and this is true also of many text-books. It is a well-known fact that after death the number of mitoses found in a given tissue decreases with time, and it is therefore not surprising that in tissues fixed many hours after death the number of mitoses found may be small, even in rapidly growing tissues. Furthermore, many mitoses become indistinct and it is difficult to distinguish them from the nuclei of degenerating cells. In the authors' cases the autopsies were performed a very short time after death, thin slices of tissue were fixed and rapidly penetrating fixatives were used. They believe that these factors enabled them to find the mitotic divisions. When they compared sections of other organs from their autopsy material, 50% of which was fixed less than one hour after death, with those from other laboratories in which they had worked and where the interval between death and autopsy averaged more than twenty-four hours, the difference in the number of mitotic divisions was striking. The same is true of surgical material instantly cut into thin slices and fixed after removal from the body as compared with large specimens, often whole organs or large tumours, when left untouched for some time or placed in their entirety into a fixative. A brief description of one case of acute glomerulonephritis, one case of subacute glomerulonephritis and one case of malignant sclerosis is given. In the first case mitotic divisions were found in the endothelial cells of the glomeruli, in the second case in the epithelial cells of the glomeruli, in the third case in both endothelial and epithelial cells.

The Synovial Membrane in Charcot's Joint.

ACCORDING to E. S. J. King (*Archives of Pathology*, June, 1941), most of the descriptions of diseases of joints include mainly the more gross and obvious alterations in bone, cartilage, ligaments and other tissues and give little account of the synovial membrane. This paucity of information is especially apparent in the case of Charcot's disease of joints, and since in this condition certain changes are more striking than in many other diseases of joints, they are described in this paper. In order to demonstrate the finer changes in cells, special attention has been given to the protoplasmic "organoids", especially the Golgi apparatus. In most of his cases the author found an increase in the amount of synovial membrane in the joint. This is, according to him, due to two factors: transudation from the blood vessels (comparable with œdema of other parts of the body) and an actual increase in the mucinous material, this being associated with an increase in the number of cells. These cells, even when desquamated, had an amazingly well-developed Golgi apparatus; often it was enlarged and occupied a considerable part of the protoplasm. These and other observations lead the author to regard the synovial fluid as fluid tissue rather than as a secretion of the lining cells of the synovial membrane, and he sums up his reasons as follows: (i) it contains cells which are morphologically and functionally normal; (ii) changes occur in the fluid which are most readily explained on the assumption that the fluid is under definite cellular control; (iii) in abnormal conditions, especially if the tissues are very active, the synovial membrane may be shown to merge into the fluid. Synovial fluid thus may be regarded as a connective tissue with a very fluid matrix, and from the point of view of the physical character of intercellular matrix, the various connective tissues exhibit a gradation from bone at one extreme, through cartilage and the various forms of fibrous connective tissue and mucoid tissue, to synovial fluid at the other end.

Villonodular Synovitis.

UNDER the heading "Villonodular Synovitis, Bursitis and Tenosynovitis", H. L. Jaffe, L. Lichtenstein and C. J. Sutro (*Archives of Pathology*, June, 1941) have developed a conception of the linkage between the tenosynovial lesion that has commonly been denoted xanthoma, xanthogranuloma, giant-cell tumour or myeloplaxoma of the tendon sheath, and the synovial and bursal lesions, likewise often classified under such heads but also in certain forms (at least so far as the synovial membrane is concerned) under other headings, namely, "chronic hæmorrhagic villous synovitis", "giant-cell fibrohæmangioma", "fibrohæmosideric sarcoma", "sarcoma fusogigantocellulare", and "benign polymorphocellular tumour of the synovial membrane". In nine of 20 cases of involvement of the synovial membrane there was a circumscribed affection consisting of one or more yellow-brown sessile or stalked tumour-like nodular outgrowths of the type commonly referred to as xanthoma, giant-cell tumour and so on. In eleven cases the involvement was diffuse and the affected synovial membrane was brownish and appeared bearded, with delicate, though somewhat matted, villi, and contained villi intermingled with numerous coarse nodular outgrowths or presented an even more complex pattern through intergrowth of villi or of villi and nodules. It is to such diffuse synovial lesions that the names noted, other than "xanthoma" or "giant-cell tumour", have often been applied. The authors' experience with the bursal lesion comprises four instances of diffuse pigmented villonodular involvement. Among 55 instances of so-called xanthoma, giant-cell tumour *et cetera* of tendon sheaths were several that shed light on the evolution of these lesions and that in this respect clearly linked them with villonodular synovitis and bursitis. As to the causation and nature of the condition, the authors reject, largely on the basis of the cytological findings, the interpretation of the lesion as a true tumour and favour the theory that the condition represents an inflammatory response to an unknown agent. From the clinical point of view, emphasis is laid on the predilection of pigmented villonodular synovitis for the knee joint and of pigmented villonodular tenosynovitis for the region between the wrist and the finger tips. As a diagnostic criterion of diffuse villonodular synovitis of the knee joint, emphasis is laid on the presence of serosanguineous fluid on aspiration, and especially on repeated aspiration. As to treatment of the lesion in any site, surgical excision is stressed as appropriate. Notice is also taken of the value of X-ray therapy in the prevention of or treatment of recurrences, particularly those of diffuse lesions of synovial membranes, but also recurrences at other sites when the lesions cannot be, or have not been, completely removed.

Metastatic Tumours of the Myocardium.

GORTON RITCHIE (*The American Journal of Pathology*, July, 1941) publishes an analysis of a series of 16 cases of metastatic tumours of the myocardium found in the course of 3,000 autopsies. He shows that there is great variety in type and origin of the primary lesions; in 16 cases 13 different types of primary tumours are represented. There were three examples of carcinoma of the lung and two of carcinoma of the pancreas; but these were the only types to be repeated. This is in agreement with statements in the literature that metastasis to the heart occurs from neoplasms of all the main organs. On further examination it appeared that seven of these 16 primary tumours were located in or about the thorax. This observation could logically be anticipated, since such tumours are usually in fairly close proximity to the heart. It indicates a possibility of regional lymphatic dissemination which should be borne in mind when the clinical course suggests cardiac metastasis. In 10 of the 16 cases the distribution of metastases could be considered as generalized. On the other hand, in one case, a carcinoma of the œsophagus, the myocardium was the site of the only remote metastasis, the bronchi and mediastinum being invaded by direct extension. Three routes of invasion are recognized, namely, the blood stream, the lymphatics and direct extension, lymphatic invasion being from the mediastinal lymph nodes against the lymph stream. In the group presented all three modes had occurred. A combination of routes was followed in several cases, malignant cells being transported through the blood stream to the epicardium, passing thence into the muscle through the lymphatics or by direct extension. In certain cases in which extensive invasion had occurred, it was impossible to determine with certainty the route of metastasis by examination of microscopic sections. Again, in a few cases, malignant cells were seen in both the blood vessels and lymphatics of the myocardium. Whether the cells in the

two vascular systems had been transported independently or whether this invasion represented the rupture of tumour cells into vessels within the myocardium could not be determined. The modes of growth within the muscle, as distinct from the routes of metastasis, were those characteristic of the various types of tumours and took three general forms: (i) through the channels, such as the lymphatics and blood vessels; (ii) infiltrative invasion with varying degrees of myocardial destruction; (iii) massive growth (microscopically speaking) with displacement and complete destruction of muscle fibres. No primary tumours of the heart were found. Consideration of the ages of the patients revealed nothing of particular interest, as in general they corresponded to the age groups in which these types of tumours are apt to be found. Twelve out of the 16 patients were males. This appears to represent a great predominance in the male sex; but such a conclusion is modified when it is considered that in 857 cases of malignancy in the series studied there were 594 males and 263 females, a proportion not far from that found in the series of myocardial metastases.

Observations on Autohæmolysis in Familial Acholuric Jaundice.

J. V. DACIE (*The Journal of Pathology and Bacteriology*, May, 1941) deals with the demonstration of autohæmolysis *in vitro* in a series of 13 cases of familial acholuric jaundice and two patients with acute hæmolytic anæmia. He found that the erythrocytes in familial acholuric jaundice underwent hæmolysis more rapidly than normal cells when kept *in vitro* at 37° C. Lysis can sometimes be seen on incubation for five hours and is often well marked on incubation for twenty-four hours. The rate of autohæmolysis is reduced in the absence of plasma or serum, but is nevertheless more rapid than that of washed normal cells. It is independent of the presence of complement and is not inhibited by normal plasma. The increased rate of hæmolysis appears to depend upon an abnormality of the erythrocytes, possibly due to increased adsorption of lysolecithin *in vivo*.

MORPHOLOGY.

Dorsal Arteries of the Foot.

J. F. HUBER (*The Anatomical Record*, July, 1941) has investigated the arterial pattern on the dorsum of the human foot. In only a little over 5% of 200 cases did he find the "standard" pattern. The anterior medial and lateral malleolar arteries arise more frequently from the *dorsalis pedis* than from the anterior tibial artery. Any one of five vessels might fill the role of "anterior perforating artery". In about 50% of cases a hitherto undescribed branch of the anterior tibial artery either contributed to, or was the principal source of, the "perforating branch of the peroneal artery". There is a high degree of bilateral similarity between the two feet. The foot of the Negro conforms more closely to the text-book picture than does that of the white.

Lymphatics in Omental Adhesions.

P. H. SIMER AND R. L. WEBB (*The Anatomical Record*, June, 1941) record the formation of new lymphatic channels in adhesions experimentally established between the omental bursa and the jejunum in dogs. Seventeen experiments were performed; the proximal end of the jejunum was used and its blood vessels were ligated. The animals were left for periods ranging from six to 245 days. In three cases left over 80 days the adhesion had broken down. Lymphatic channels were traced by feeding with fat just prior to sacrifice of the animal. Ordinary fat revealed no new channels; fat stained with Sudan III of a particular make gave no positive findings. Sudan IV of another make was then employed and stained fat could be seen in the mesenteric lymphatics; in two of the six dogs used for this experiment the coloration could be traced to the omental and duodenal lymph nodes. Microscopic examination of sections cut through the region of the adhesion confirmed the presence of new channels connecting the intestinal lymphatics with omental lymphatics.

The Visual System in the Phalanger.

A. D. PACKER (*The Journal of Anatomy*, April, 1941) reports the results of experimental investigations on the visual apparatus of *Trichosurus vulpecula*. All fibres of the optic nerve are myelinated; about 75% of them cross in the chiasma. Crossed visual fibres pass to the dorsal and ventral nuclei of the lateral geniculate body, to the *corpus quadrigeminum superius* and to the *nucleus opticus tegmenti*. Uncrossed fibres were traced only to the dorsal nucleus of the geniculate body. In this nucleus crossed and uncrossed fibres end chiefly in alternate cell laminæ. The ventral quarter of the geniculate body is poorly myelinated and receives only crossed fibres. Corticipetal fibres from the geniculate body end chiefly or entirely in the *area striata* in reversed representation; corticifugal fibres were traced to the dorsal nucleus of the lateral geniculate body and to the *corpus quadrigeminum superius*.

The Urachus.

G. HAMMOND, L. YGLESIAS AND J. E. DAVIS (*The Anatomical Record*, July, 1941) have studied the urachus and its relations in 35 approximately full-term fœtuses, one three-year-old infant and 100 adults. In the fœtus the conical bladder dome leads through a gradually diminishing urachal canal to the umbilicus. The umbilical arteries ascend independently to end close to the urachus in the umbilical scar. The urachus has its own artery (from the superior vesical artery) which effects anastomoses with the umbilical arteries. The urachal musculature is well developed and continuous with that of the bladder. In 50% of cases the lumen of the urachus was continuous with that of the bladder by an opening less than one millimetre in diameter; in no case could a valve of Wutz be found. Where there was no continuity of the lumina the urachal epithelium could usually be traced through the vesical musculature; but sometimes this intervened. Frequently a meso-urachus could be found, extending from the umbilicus to the *symphysis pubis*. The adult form of the urachus is affected by relative amounts of growth and atrophy, and there is great variation. One series showed the urachus as a distinct cord from the bladder almost to the umbilicus, averaging 12 centimetres in length; in one specimen the lumen communicated with that of the bladder, and there was a meso-urachus. In another series the urachal cord joined one of the obliterated umbilical arteries at an average distance of 6·4 centimetres from the bladder; in one case there was communication between the urachal lumen and that of the bladder. In a third series the urachal cord and the umbilical arterial cords converged at a point about midway to the umbilicus. The fourth group contained specimens with the shortest urachal lumen. In these the urachus averaged 3·4 centimetres in length. In about one-quarter of the cases the urachus was a muscular tube, in another quarter only a fibrous cord; in the remaining cases it was a muscular tube tapering to a fibrous cord in its upper half. When present, the urachal canal was about one millimetre in diameter and was lined with epithelium; in only two cases did the lumen enter the bladder cavity. In relation to the urachus are two fascial planes. One, the umbilical prevesical fascia, is derived from the peritoneum which originally invested the superior part of the bladder, the urachus and the umbilical arteries in a mesentery-like fold. This fascial layer is triangular, with the apex at the umbilicus; the lateral margins blend with the extraperitoneal tissue along the obliterated umbilical arteries, the lower extends behind the pubis to join the fascia covering the *levatores ani*. The other fascial plane, the umbilical vesical fascia, is also triangular with the apex at the umbilicus. It lies between the umbilical prevesical fascia and the parietal peritoneum, and invests the urachus, obliterated umbilical arteries, bladder and prostate, and the related nerves, blood vessels and lymphatics. The authors point to the clinical importance of these fascial planes.

Renal Fascia.

E. G. CONGDON AND J. N. EDSON (*The Anatomical Record*, July, 1941) have studied the renal fascial layers in a series of one-inch cross-sections in 74 specimens. Only two renal fasciæ occur in relation to each kidney: the anterior renal fascia (of Gerota) and the posterior renal fascia (retro-renal fascia of Zuckerkandl). Contrary to general belief, not more than half of each kidney is covered by renal fascia. The perinephric *panniculus adiposus* which lies between the kidney and the renal fascia is thus bare above where it is called the epinephric *panniculus adiposus*. The anterior and posterior renal fasciæ, together with the fasciæ on the *psoas major* and *quadratus lumborum*, fuse below to form an inverted fascial cone which invests the inferior pole of the kidney. Another fascial layer, called the lateroconal fascia, extends laterally from this cone, behind the colon, to join connective tissue of the peritoneum in the paracolic gutter. The anterior renal fascia is stronger than the posterior, ending above usually at the level of the upper half of the body of the second lumbar vertebra; below, it joins the psoas fascia, the psoas muscle or the quadratus fascia. The anterior and posterior renal fasciæ never unite over the upper pole of the kidney, nor is there a fascial layer between the kidney and suprarenal. Above, the lateroconal fascia gradually disappears, then the lateral border of the anterior renal fascia fuses with the peritoneum. Medially, the renal fascia is not continuous across the mid-line with that of the opposite side. In no case did the ureter pierce the renal fascia. The cone of renal fascia can be demonstrated radiologically after injection of air into the perinephric panniculus.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on September 3, 1941, at the Medical Society Hall, East Melbourne, DR. H. BOYD GRAHAM, D.S.O., M.C., the Acting President, in the chair.

Industrial Fatigue.

DR. H. M. L. MURRAY, prinicpal medical officer to the Ministry of Munitions, read a paper entitled "Industrial Fatigue" (see page 437). At the outset Dr. Murray made it clear that he was about to express his own views, which were not necessarily those of the Ministry of Munitions, and that he was not speaking in an official capacity.

DR. GUY SPRINGTHORPE said that Dr. Murray was to be congratulated on bringing the subject of industrial fatigue before members of the Branch. At the present time it was a question of national importance. The paper was so clear and convincing that little could be added to it. The causes of industrial fatigue fell into two main classes: first, there were the general environmental factors governing working conditions and affecting all workers alike, and secondly, there were personal factors peculiar to each individual. These latter included both the physical and the mental side of each worker's health. However, until optimum general conditions of work suited to persons of average ability and health were provided, little benefit would follow from a detailed consideration of individual difficulties and differences. Under the conditions at present prevailing amongst war workers in Australia, Dr. Murray had rightly confined his remarks to the fundamental general factors affecting output. Members of the medical profession were not only interested in the efficiency of the war effort, but were also concerned with the health of the community. Dr. Springthorpe said he would like to ask Dr. Murray whether, as a result of the unscientific working conditions referred to, he had noticed any increase in illness amongst the operatives resulting from over-fatigue. From conversations with medical colleagues, he had concluded that such illness was becoming increasingly prevalent in general practice. Recently the National Fitness Council of Victoria, after conferring with Dr. Murray, had adopted a statement which it was proposed to send out as a circular to employers. This Dr. Springthorpe proceeded to quote, as follows:

> Capacity for work is governed by the fitness of the individual.
>
> To achieve the best results, absence of fatigue, both mental and physical, as well as normal health, is essential.
>
> Conditions of work affect health and may cause fatigue if:
>
> 1. Hours are too long.
> 2. Rest and meal intervals are inadequate.
> 3. There is no full day off each week.
> 4. There is not a full fortnight holiday per year.
>
> It was proved in England during the last war and rediscovered in this war that when these prerequisites are not fulfilled both quantity and quality of work deteriorate.
>
> So far during the present war effort in Australia these fundamentals have not been fully recognized. The National Fitness Council urgently directs the attention of all employers and employees to these vital facts.

Dr. Springthorpe went on to say that it appeared that not only in war industry, but also in many civil occupations, the four prerequisites referred to were not always provided. If, as appeared likely, the health of many workers would be endangered by any long continuation of the present inefficient methods, it might well be desirable for the British Medical Association, either in its State or Federal capacity, again to approach the administrative authorities in an effort to have conditions radically improved, and in this way give support to Dr. Murray, who so far, on his own admission, had been but a voice crying in the wilderness.

DR. GERALD WEIGALL asked Dr. Murray whether the figures which he had quoted from Vernon's tables relating to output referred only to perfect articles or whether rejects had been included. Dr. Weigall wondered whether the factor of fatigue tended to increase the proportion of faulty work and consequent wastage of material.

DR. H. F. MAUDSLEY, after expressing his appreciation of the paper that had been read, asked Dr. Murray whether he had realized that, though in the types of fatigue problems with which he was confronted fatigue was a negative quality, the negative symptoms produced positive effects, many of which would be devastating. Dr. Maudsley had been struck with the types of patients he met in psychiatric clinics who were able to carry on work. As it was a fact that the army absorbed many of the fit people, many of the unfit must drift into munition work, and amongst munition workers they must find all sorts of queer people. It was a horrifying thought that men of that type might be in positions where they might do great harm. Dr. Maudsley wondered whether psychiatric problems showed up at all prominently under the stress of fatigue.

DR. J. NEWMAN MORRIS said that he wished to refer to Dr. Murray and his work. It was apparent to all that Dr. Murray's knowledge, continued study and enthusiasm formed a refutation of the argument sometimes advanced that a salaried officer tended to lose interest in his work. The country was to be congratulated on having such a keen officer in so important a job, and it was disappointing that he felt that he had not had an adequate response from the constituted authorities. He had presented his evidence and his advice to industrialists, politicians and health authorities without making satisfactory headway. Attention had been drawn to the same problem by evidence placed before the Royal Commission on Health in 1926, and soon afterwards the Department of Health had established an industrial section; but it had ceased to function early in the days of the depression. It had never been revived, though the Federal Council of the British Medical Association had approached the Commonwealth Government on the matter. Dr. Morris thought that as a Branch they should back Dr. Murray and become vocal on matters of health reform. He mentioned as an example of successful adoption by the British Medical Association of a strong attitude in health matters the improvement that had followed representations in the matter of collection and distribution of milk. If Dr. Murray could obtain support in the right quarters, he would not have spent his efforts in vain.

DR. H. L. STOKES said that as Dr. Murray and Dr. Springthorpe had each referred to the break for lunch, he wondered what the optimal length of that break should be and whether the meals available for the operatives should be hot. He also asked whether Dr. Murray was prepared to advocate the introduction of intervals of five minutes each in the middle of the morning or the middle of the afternoon to interrupt the monotony of the work; whether music was provided or anything was done to alleviate boredom; and whether the work from which he had demonstrated his thesis could be classified as piece work or day work.

DR. C. H. DICKSON asked Dr. Murray whether he had made his appeal to the authorities on the basis of saving money; a ten-hour shift would mean paying for only two hours' overtime, whereas a twelve-hour shift involved payment for four hours' overtime, apparently without any increase in output for the extra money.

DR. H. BOYD GRAHAM, from the chair, thanked Dr. Murray for his lucid, complete and satisfactory paper, and for having brought so important a matter to the notice of the members of the Branch. He asked Dr. Murray whether any consideration had been given to the question of the spare-time occupation of the operatives, and whether any experiment had been made on the basis of short shifts with short intervals between shifts for the operatives concerned. He mentioned active service conditions in the collection and dressing of casualties. Under the imperative stimulus of collection of wounded soldiers no rest or sleep had been obtained for seventy-two hours on one occasion; that period was followed by twenty-four hours of uninterrupted slumber and restoration of normal efficiency. With a never-ceasing supply of wounded arriving at a dressing station, by trial and error it was found that shifts of six hours on and six hours off could be maintained for as long as nineteen days. In the time off, meals were taken, shaving and personal hygiene were attended to, and sleep was obtained. For work of urgent national importance, such as the making of munitions in time of war, he considered that at least some of the operatives should be attested persons with service control over the full twenty-four hour cycle.

Dr. Murray, in reply, said that Dr. Springthorpe had asked for information about loss of hours amongst operatives from increased incidence of illness; though he had no exact figures, he felt that it was the case that the long shifts were associated with loss of time owing to ill health. The records at Lithgow showed a steady increase in the average number of days of sick leave during the three years in which twelve-hour shifts had been worked at the factory. Dr. Murray said that Dr. Gerald Weigall had asked an awkward question. He could not recall any reference in Vernon's work which would enable him to say whether he

had made his calculations only on perfected articles; but he thought from the nature of the work and the machines it would be almost impossible to produce an imperfect article. He informed Dr. Maudsley that, though it was probable that workers with a psychiatric trend would develop symptoms of imbalance during employment in proportion to the stress of the work, he had concentrated his attention mainly on the aspects of efficiency and output. Old operatives when interrogated had informed Dr. Murray that they did not know much about what was happening in the last three or four hours of a twelve-hour shift. It was necessary to depend on the cooperation of foremen and supervisors to obtain reports indicating that a particular operative was unable to maintain efficiency, and then that operative was examined and if necessary sick leave was obtained for him. He remembered a man who had said very confidentially that he was engaged on special work known to only two people, when in fact some five hundred workers were engaged on the same work in that shop. In reply to Dr. Stokes, Dr. Murray said that he had already suggested that the optimal time for meals should be of the order of one hour; but in numerous workshops only twenty minutes were allowed from cessation to resumption of work, and in that time distances up to a quarter of a mile to and fro might have to be travelled from the workshop to where the meal was taken. It was due to the faulty arbitration awards and the legal and secretarial training of those who drafted the regulations that a vicious system had been evolved with the object of packing a full eight hours of work into an eight-hour shift with as little time as possible for meals. Whether the meals were hot or cold depended on individuals, and little was done about the question in government factories. With reference to rest pauses, Dr. Murray said that attempts had been made to have breaks of five minutes each introduced into four-hour shifts; but the lay officers had refused to allow them. He had obtained no specific figures bearing on the relationship between boredom and fatigue. The examples he had used from Vernon's work related to piece work; but he had quoted two examples illustrating that the effect of reduction of hours was approximately the same for piece workers as for day workers. Work just started at Lithgow also indicated similar results. In reply to Dr. Dickson, Dr. Murray said that he had frequently used the argument of less payment for overtime; but even that approach had not met with success, so Dr. Murray had concentrated on pressing the aspect of efficiency of production. In reply to Dr. Graham, Dr. Murray said that on the medical side there was no direct concern with what the operatives did when they were not working; but there was a welfare department which was attempting to improve matters, though under present conditions of transport and with the workers' homes scattered all over the city, it was difficult to do anything effective. More than half the working population lived more than five miles from the factories, and in the special case of Lithgow the operatives lived about thirty-five miles away. At Lithgow they had the maximal sickness absenteeism; if a man had a cold or a boil he was likely to be away from work for a week because he was tired out. In existing circumstances it was not possible to have shorter shifts and shorter intervals for each worker between shifts. In conclusion, Dr. Murray thanked the audience for the patient hearing he had received.

Special Correspondence.

CANADIAN LETTER.

From Our Special Correspondent.

Our annual meeting this year was held in Winnipeg. We follow the plan of moving from one end of the continent to the other. Next year the meeting will be in Alberta, and then probably in British Columbia, then back to the east. We have not yet reached the stature of the American Medical Association, which is now so colossal that only a few cities are capable of handling its annual meeting. We usually manage to give every province its turn.

Our Winnipeg meeting was notable for the excellence of its morale, but naturally the times have left their mark on our numbers. Many members of the Association of course are in uniform. One of them was the Director-General of Medical Services of the Canadian Army, Brigadier Gorssline, who attended the meeting and gave an excellent résumé of the development and work of the medical department of the army, the Royal Canadian Army Medical Corps. It is always interesting, and in this case encouraging, to take stock of growth and performances. One is always impressed by what has been done in so short a time. There has not been the dislocation of medical services for the civilian population which was feared, but it must be admitted that many hospitals and communities feel the effects of the gradual draining away of medical staff. In my own hospital, the Montreal General Hospital, no less than 75 members of the staff are on active service. This includes numbers of the house surgeons; but that does not make it any easier for the remaining skeleton to carry on the same amount of work which faced it two years ago. Rightly or wrongly—for there are two sides to it—a great many of the older men have also joined, and it is felt that some of them, experienced veterans though they might be, were not able to stand the strain of active service. But of course they filled a gap when they were most needed, and possibly there will be some shifting and exchange of men in favour of younger men. Already we have had men back from England who have brought back the latest methods of medical work in the field.

Brigadier Gorssline showed that the medical officers of the Royal Canadian Army Medical Corps now totalled over one thousand, and nursing sisters nearly seven hundred. He commented on the introduction of X rays into the regular examination of recruits. Up to date well over 300,000 men had been X rayed, and of these 1·6% had been rejected. About 58% of those rejected were shown to have pulmonary tuberculosis, and 21% other lung disease. As regards immunization, the most notable advance has been the development of a combined "TAB" vaccine with tetanus toxoid.

The question of medical teaching is acute. There was some discussion this year as to the carrying on of teaching throughout the summer, so as to shorten the length of the course, but for this session it was not found feasible, at least in all the schools. Classes are being held throughout the summer in Western University, London, Ontario; the men will therefore be able to graduate about three months earlier than usual. In McGill, however, a four-year course had already been in operation before the war, after about twenty-five years of the five-year arrangement. The change was made largely on economic grounds, and called for a great deal of work at the beginning. Now, however, its advantage is being generally admitted under the persuasive influence of the war, and Toronto, the largest Canadian medical school, is aiming at it. The poor medical student is feeling rather more compressed than usual, as he has to get in some military training along with his medical work.

No city in Canada has felt the impact of the war with greater force than Halifax, Nova Scotia. This has reflected itself in certain medical aspects. Halifax, with all its fine seaport facilities, had up to the time of the war, never been more than moderately busy, and then chiefly in the winter, when the St. Lawrence shipping had closed down. With the war, Halifax became not only busier than it ever had been, even in the last war, but its population swelled almost incredibly, for the amount of housing available. It is now credibly reported that, far from there being any difficulty in renting rooms or houses, people have in some places to display cards to the effect that "this house is not for rent".

This influx of people led not only to crowding, but to the bringing together of men from all countries. Consequently, infectious diseases had a great chance. Unfortunately, too, Halifax had allowed its public health preventive measures to slacken somewhat, particularly in regard to diphtheria inoculation. The result was that the medical department of the city was swamped with work for a time. However, as one of the very few gains to be credited to the war, civic expenditures for public health were considerably increased to something corresponding with what they should have been, and inoculation for diphtheria was gradually put in force, but only after extraordinary exertions on the part of the medical officers of the city.

Correspondence.

WHO GROWS THE CABBAGES?

Sir: The complacency of our councils, alleged by some recent correspondents, is, to my mind, real. The councils are, however, not conscious of their frame of mind, and undoubtedly think that they are giving their best for the Association, and do spend a deal of time on matters to our general interest.

The complacency is the result of a constitution and a set of rules that are impracticable and out of date. The average member has no idea nor any means of finding out the fitness of any nominee to the council to hold the position.

But at the annual ballot the same old names, with an odd new one, come along to each member so that he can express an opinion. What opinion can he express? He can only say that one is more of a good fellow than the other. He is not expressing an opinion on the policy. At the ballot we are restricted to crossing off those few nominees that are numerically superfluous. If we cross off more, our vote, such as it is, is not allowed. At every ballot I would like to put my pen through several extra names.

The complacency of our councils is added to by the manner in which important matters of policy are dealt with by the so-called general meetings. After due notice of motion is given, all members are circularized and notified of date of meeting and the notice of motion. But the point is that the large majority of our members are prevented by time, distance and the exigencies of our calling to attend such a meeting, and actually have no vote at all in the matter.

As at present constituted the councils have no proper democratic right to work out new schemes of practice nor to enter into any conference with government departments as regards our interests in any national insurance schemes. But they have a big urgent job if they desire to weld us into an organization which will give them that real right to represent us.

It is up to the various councils to wake the members from their apathy and give them the chance to become vocal. Let us have a new method of electing our council, something akin to the suggestion of Dr. Walker, of Western Australia (THE MEDICAL JOURNAL OF AUSTRALIA, August 30, 1941), where each State is divided into electorates with a representative of each. Then the country members, and those members in big towns other than the capitals would soon find amongst the city or suburban practitioners a representative who knew their own particular problems. And let all matters of policy, that the council decide should be put to the vote, be voted on by all members, by means of the postal service.

If some such reorganization was commenced, the then council would have no cause to complain of the apathy of members. This apathy is the fruit of the councils' complacency.

Yours, etc.,

ALAN PITT.

Cunnamulla,
Queensland,
September 19, 1941.

SIR: I welcome Dr. Pitt's criticisms as evidence of an awakening interest in the affairs of the Branch. I agree with him that it is a matter of vital importance to the Branch that members should freely express their views and assist in the decisions that the council is called upon to make.

In charging the council of the Queensland Branch with complacency, and in implying that no consideration is given to the point of view of extra-metropolitan practitioners, Dr. Pitt displays an ignorance which is excusable in one who, like "Rip Van Winkle", has awakened from a long sleep.

The facts are:

1. In 1928 a member of the council was chosen to represent each particular district. This proved to be of no value and ceased to operate.

2. On June 11, 1937, the council resolved that for a period of one year four country members be coopted without any alteration in the by-laws and that the desirability of amending the by-laws be reconsidered by the council in August, 1938. As a result the following appointments were made to the council on the nomination of the local associations:

(i) Northern—Townsville and Cairns: Dr. V. F. A. O'Neill.
(ii) Central—Rockhampton and Hinterland: no appointment was made.
(iii) Gladstone, Bundaberg, Maryborough, Gympie and Kingaroy: Dr. G. R. Woodhead.
(iv) South-western Queensland—Dalby to Charleville, Goondiwindi and St. George: Dr. A. W. Fox.

This method of representation met with no success.

3. In November, 1938, the council decided that representatives of the local associations be invited to attend annual conferences with the Branch council, and the by-laws were altered accordingly (*vide* By-Law 69, page 115, "Handbook for Qualified Medical Practitioners", issued by the British Medical Agency of Queensland, Proprietary, Limited). These conferences, which have been held in 1939, 1940 and 1941, promise to be of value; but the war has interfered with the ability of representatives to attend them.

4. A standing invitation is given to members to attend council meetings when possible.

5. Four members of the present council represent extra-metropolitan areas. In addition, one has recently arrived in Brisbane after years of country practice and two have had some years of experience in country practice.

6. Whenever possible, all matters affecting the interests of the Branch are referred to local associations and to members by post. At the moment a ballot is being conducted concerning a proposal to establish a medical benevolent fund.

In conclusion, nomination papers for the 1941 elections have just been issued, and I hope that many members, metropolitan and extra-metropolitan, will find time to make a nomination to the council on the form provided.

Yours, etc.,

J. G. WAGNER,
President.

British Medical Association House,
225, Wickham Terrace,
Brisbane.
October 2, 1941.

REFUGEE DOCTORS.

SIR: In the correspondence in regard to the registration of refugee doctors, sufficient red herrings have been drawn across the path of discussion to reduce the price of fish here to a most reasonable level.

I feel sure that most of these refugee doctors are men of at least normal intelligence. All Australian graduates realize that after graduation they will only be able to practise their profession within the British Empire. This was so pre-war; if they desired to practise in foreign countries they were obliged to pass the necessary medical examinations in those countries and/or become citizens of that country. I doubt whether any of the alien or once alien practitioners were unaware of the fact that Australian registration boards reciprocated accordingly. Consequently, if these refugee doctors desire to practise here as registered medical practitioners, all they are required to do is to pass the necessary and required medical examinations (examinations conducted by the university); recently three foreign graduates adopted this legal method. And whilst they are doing this to conduct themselves according to our ethical principles and not display plates labelled "Dr. ——, not registered in New South Wales".

Yours, etc.,

C. H. JAEDE.

4, Robey Street,
Mascot,
New South Wales.
October 3, 1941.

SIR: Dr. Roseby's letter criticizing my views on the above subject goes into questions that I never raised.

The burden of my letter is the clamour in the Press, in this State, at any rate, for the registration of all and sundry alien doctors. I have no doubt that their pass qualification entitling them to registration in their own country is of a lower standard than that of Australian universities, and which standard I hope will never be reduced.

It might be as well to call Dr. Roseby's attention to the proceedings of the tenth session of the National Health and Medical Research Council, held at Canberra on May 28 and 29, 1941, from which I extract the following, as reported in THE MEDICAL JOURNAL OF AUSTRALIA of August 16, 1941, Volume II, Number 7, pages 174 and 175:

> Under the heading "Public Health under War Conditions" several important matters were considered. The question of the registration and employment of alien medical practitioners was discussed and the following resolution was adopted:
>
> That, as far as can be ascertained, there are less than one hundred doctors with foreign qualifications who are not registrable under existing State laws.
>
> That, because of some Continental systems of training, a large proportion cannot be considered as possessing sufficient knowledge or experience to undertake general practice under Australian conditions. Such persons should not be allowed to practise in remote localities.
>
> It is considered probable that, after the war, a considerable influx of such doctors into Australia will occur, and, therefore, the situation which will thus arise should be provided for by anticipatory action.
>
> Such action consists in amending existing law where necessary by:
>
> (a) prohibiting the practice of medicine for profit by unregistered persons;

(b) by giving medical boards discretionary powers to certify that the course of training undergone by, and the knowledge and experience of, the applicant is equivalent to the standard required by Australian universities—and upon the issue of such certificate to register the applicant;

(c) continuing to offer to all alien graduates special university and hospital courses of at least three years' duration and the opportunity of passing Australian qualifying examinations.

That the most satisfactory method of meeting the conditions in the sparsely settled districts is the provision of a whole-time medical service under the *Public Service Act* with appropriate and adequate official transport.

The necessity for aliens to do the last three years of the medical course and pass the prescribed examinations would be some protection to our standards in Australia.

It does seem a pity that the personal element should be brought into such a discussion; I never questioned the high standing of the many eminent men in their various specialties in all parts of the world; *vice versa*, perhaps Dr. Roseby will even admit the translation of eminent Australian anatomists to the Old World and the influence of Australians in original work on prostatic surgery and the autonomic nervous system.

As to higher degrees, it is well known that a Paris M.D. may be obtained and it will not allow the recipient to practise in France—there is one alien doctor in Sydney now with a Central European degree and such an M.D. Paris. The only calendar of the University of Paris that I can consult in Sydney is *"Le Livre de l'Université de Paris"*, 1929-1930 copy, and on page 73 it states that the doctorate of the University of Paris does not confer the right to foreign students to practise medicine in France. Before doing so they require a Diploma of State, which necessitates being naturalized, further study and further examinations.

I cannot find a copy of the calendar of the University of Berlin or of Vienna; possibly there, also, may be similar restrictions.

Yours, etc.,

T. W. Lipscomb.

135, Macquarie Street,
Sydney,
October 7, 1941.

Naval, Military and Air Force.

APPOINTMENTS.

The undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 197, of October 2, 1941.

Australian Military Forces.

Australian Army Medical Corps.

Captain (provisionally) (Temporary Major) J. R. Nimmo is reappointed for a further period of two years commencing 6th November, 1941.

Seventh Military District.

To be Major (temporarily).—Captain (provisionally) N107904 J. M. Wilshire, 25th August, 1941.

Northern Command.

First Military District.

Honorary Captain P. S. Woodruff is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally) with regimental seniority next after Captain (provisionally) A. J. Barnett, 19th April April, 1941.

To be Honorary Captains.—John Kenneth Mowat, 26th August, 1941; George Middleton and James Bogle, 1st September, 1941.

Eastern Command.

Second Military District.

The following officers are appointed from the Reserve of Officers (A.A.M.C.) on the dates shown: Major (now Temporary Colonel) W. Evans, M.C., and is seconded, 21st May, 1940, and Captain H. G. B. Bruce, 18th August, 1941.

The following officers are appointed from the Reserve of Officers (A.A.M.C.) on the dates shown and to be Captains (provisionally): Honorary Captains N274395 J. Gribben, 28th July, 1941, and H. B. Gatenby, 31st July, 1941.

Honorary Major L. Utz is appointed from the Reserve of Officers (A.A.M.C.) and to be Major (provisionally), 13th August, 1941.

To be Captains (provisionally).—Norman Arthur Walker, 16th July, 1940 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 198/1940, promulgated in *Commonwealth Gazette* No. 213 of 1940); and John Lindsay Taylor, 14th July, 1941 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 235/1940, promulgated in *Commonwealth Gazette* No. 1 of 1941).

To be Lieutenant-Colonel (temporarily).—Captain (Temporary Major) P. L. Jobson, 7th July, 1941.

To be Captains (provisionally).—Ernest Eric Smithers, 14th November, 1940; and James Main McDonald, 26th August, 1941.

To be Honorary Captains.—David Lindsay Graham, Murray Linton Verso, Stephen Dennis Foley, Richard Duncan Davey, 26th August, 1941; Rawdon Hamilton Kenny, 27th August, 1941; John Cranstoun McInerney and Kevin Patrick Clifford, 1st September, 1941.

Southern Command.

Third Military District.

Captain (provisionally) K. G. Brown is seconded, 1st September, 1941.

Fourth Military District.

Major E. H. Lewis, E.D., is appointed from the Reserve of Officers (A.A.M.C.), 29th April, 1940.

To be Honorary Captain.—Laurence Corin Holland, 28th August, 1941.

Sixth Military District.

To be Lieutenant (provisionally).—Leonard Arthur Cramp, 25th August, 1941.

Western Command.

Fifth Military District.

The dates of appointment of Captains (provisionally) W37 V. A. F. Stewart and W28587 J. B. Hogg, which appeared in Executive Minutes No. 96/1941 and No. 49/1941, promulgated in *Commonwealth Gazettes* No. 108 of 1941 and No. 50 of 1941, respectively, are amended to read "1st November, 1940" and "29th July, 1940", respectively.

Honorary Captains W16343 M. J. Morris and W243943 H. C. Mulcahy are appointed from the Reserve of Officers (A.A.M.C.) and to be Captains (provisionally), 8th April, 1940, and 12th July, 1940, respectively (in lieu of the notifications respecting these officers which appeared in Executive Minutes Nos. 49/1941 and 111/1941, promulgated in *Commonwealth Gazettes* Nos. 50 of 1941 and 124 of 1941, respectively).

Honorary Captain W233393 A. A. Merritt is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally), 16th June, 1940 (in lieu of the notifications respecting this officer which appeared in Executive Minutes Nos. 65/1941 and 100/1941, promulgated in *Commonwealth Gazettes* Nos. 73 of 1941 and 114 of 1941, respectively).

Honorary Captains W28584 W. A. Hillman and W28615 J. M. Flynn are appointed from the Reserve of Officers (A.A.M.C.) and to be Captains (provisionally), 13th August, 1940, and 31st August, 1940, respectively (in lieu of the notifications respecting these officers which appeared in Executive Minutes Nos. 65/1941 and 49/1941, promulgated in *Commonwealth Gazettes* Nos. 73 of 1941 and 50 of 1941, respectively).

The date of appointment of Captain (provisionally) W233392 R. G. Linton, which appeared in Executive Minute No. 49/1941, promulgated in *Commonwealth Gazette* No. 50 of 1941, is amended to read "27th May, 1940".

The following officers are appointed from the Reserve of Officers (A.A.M.C.), and to be Captains (provisionally): Honorary Captains W10 J. A. Gollan, 23rd July, 1940, and W233408 S. L. Mainland, 7th August, 1940.

Honorary Captain B. Burnside is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 9th April, 1940, and to be Major (temporarily), 21st June, 1940 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 175/1940, promulgated in *Commonwealth Gazette* No. 190 of 1940).

To be Honorary Captain.—Alexander Thomas Hicks Jolly, 26th August, 1941.

Australian Imperial Force.

Australian Army Medical Corps.

Colonel R. A. Money, M.C., E.D., and Lieutenant-Colonel J. C. Belisario are appointed to command Australian General Hospitals, 29th July, 1941.

Lieutenant-Colonel L. G. Male is appointed to command a Field Ambulance, 29th July, 1941.

Majors K. C. Purnell, M.C., E.D., and N. Eadie are appointed to command Australian Convalescent Depots, 29th July, 1941.

Major G. E. Gillespie is appointed to command a Casualty Clearing Station, 29th July, 1941.

Major K. S. Richardson is transferred from the Permanent Supernumerary List, 29th July, 1941.

CASUALTIES.

According to the casualty list received on October 13, 1941, Captain S. S. Caporn, A.A.M.C., of Gardenvale, Victoria, is reported seriously ill.

DECORATIONS.

Lieutenant-Colonel Robert Heathcote Russell, of Newcastle, New South Wales, has been created a Companion of the Distinguished Service Order.

Captain Rowland Ralph Anderson, of Perth, Western Australia, has been awarded the Military Cross.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

The undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Goodchild, Muriel Clare, M.R.C.S. (England), L.R.C.P. (London), 1935, 4, Hunter's Road, Balmoral Beach.

Friedlaender, Erich Martin Caesar, M.D., 1908 (Univ. Giessen), 49, Spit Road, Mosman. Also recommended and approved for registration in terms of section 17 (2) of the *Medical Practitioners Act*, 1938.

Lippmann, Arthur, M.D., 1907 (Univ. Kiel), 175, Macquarie Street, Sydney. Also recommended and approved for registration in terms of section 17 (2) of the *Medical Practitioners Act*, 1938.

Nominations and Elections.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Homewood, Arthur Norman, M.B., Ch.B., 1915 (Univ. Edinburgh), Imperial Service Club, Barrack Street, Sydney.

Hodgson, George Alfred, M.B., B.S., 1939 (Univ. Sydney), 135, Denison Road, Dulwich Hill.

Finlay, Donald Murray Logier, M.B., 1940 (Univ. Sydney), c.o. Naval Liaison Officer, Australia House, London.

Finlay, Sinclair Elwyn, M.B., 1940 (Univ. Sydney), c.o. Naval Liaison Officer, Australia House, London.

Kent, Merrian Winifred, M.B., B.S., 1941 (Univ. Sydney), 6, Hiawatha Flats, 63, Douglas Street, Stanmore.

Obituary.

CHARLES HENRY GEOFFREY RAMSBOTTOM.

We regret to announce the death of Dr. Charles Henry Geoffrey Ramsbottom, which occurred on September 29, 1941, at Adelaide, South Australia.

ANTONIO CHARLES HOUEN.

We regret to announce the death of Dr. Antonio Charles Houen, which occurred on October 2, 1941, at Adelaide, South Australia.

Diary for the Month.

Oct. 22.—Victorian Branch, B.M.A.: Council.
Oct. 24.—Queensland Branch, B.M.A.: Council.
Oct. 30.—New South Wales Branch, B.M.A.: Branch.
Oct. 30.—South Australian Branch, B.M.A.: Branch.
Oct. 31.—Tasmanian Branch, B.M.A.: Council.
Nov. 5.—Western Australian Branch, B.M.A.: Council.
Nov. 5.—Victorian Branch, B.M.A.: Branch.
Nov. 6.—South Australian Branch, B.M.A.: Council.
Nov. 7.—Queensland Branch, B.M.A.: Branch.
Nov. 11.—Tasmanian Branch, B.M.A.: Branch.
Nov. 14.—Queensland Branch, B.M.A.: Council.
Nov. 19.—Western Australian Branch, B.M.A.: Branch.
Nov. 26.—Victorian Branch, B.M.A.: Council.
Nov. 27.—New South Wales Branch, B.M.A.: Branch.
Nov. 27.—South Australian Branch, B.M.A.: Branch.
Nov. 28.—Queensland Branch, B.M.A.: Council.
Nov. 28.—Tasmanian Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to the Editor, The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, The Medical Journal of Australia, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

Subscription Rates.—Medical students and others not receiving The Medical Journal of Australia in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £1 for Australia and £2 5s. abroad *per annum* payable in advance.